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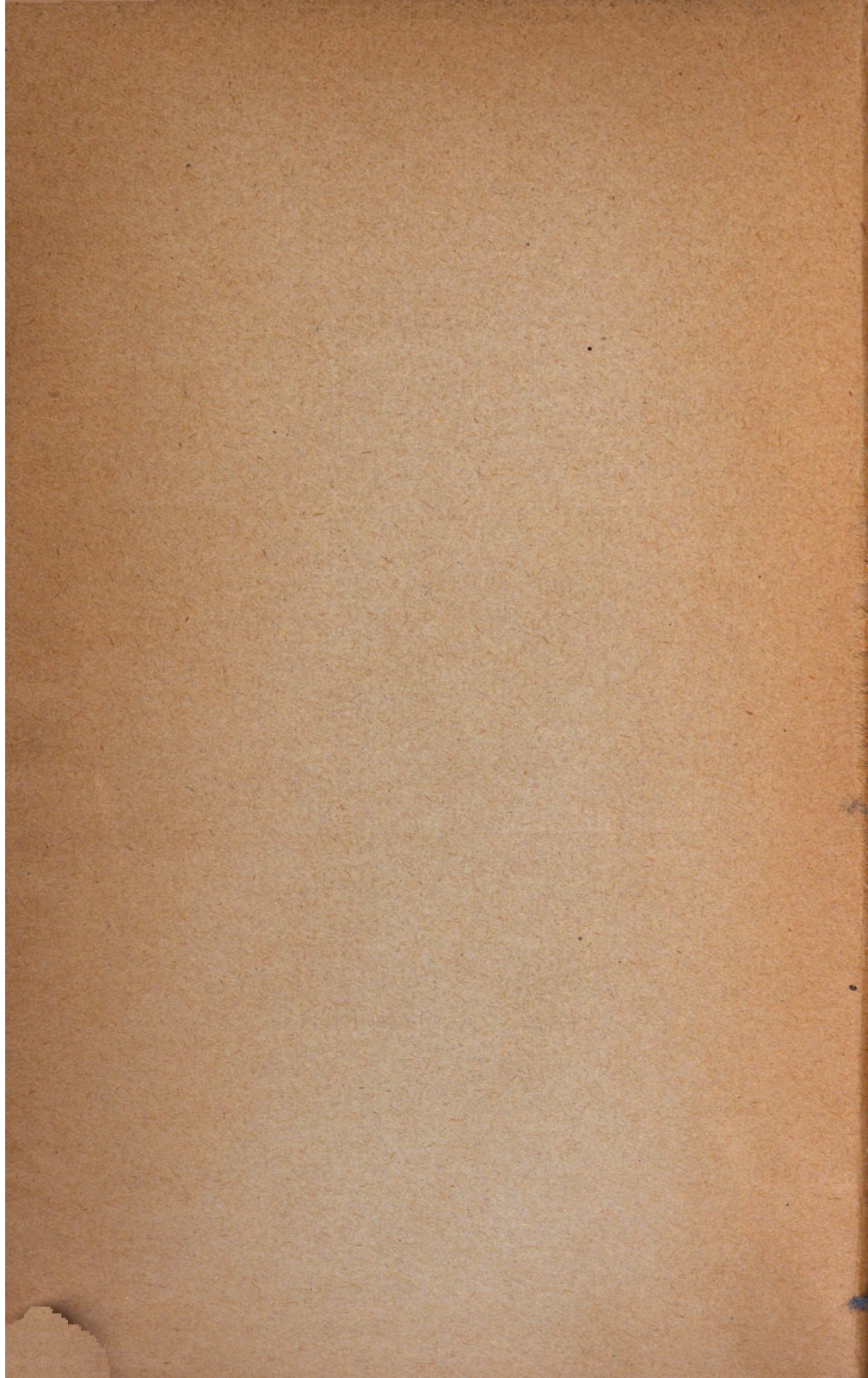
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# GUY'S HOSPITAL REPORTS.

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AND

F. J. STEWARD, M.S.

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# TWO CASES OF VOLVULUS, OF WHICH ONE WAS SUCCESSFULLY TREATED BY RESECTION OF THE TWISTED BOWEL.

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THE first of these cases was prepared for publication several months ago, but for various reasons was delayed. The occurrence of the second has led us to put them together on the present occasion.

CASE I.—Elizabeth G., æt. 43, was admitted into a medical ward on March 28th, 1899. She had always enjoyed good health until last July, when she suffered from considerable abdominal pain, constipation and vomiting, and at the same time the abdomen became very much enlarged. The constipation was absolute; the vomiting was not very frequent, and was not fæcal. The condition persisted four or five days; the bowels were at length opened by an enema, and she got well. Three months later she had a similar attack, and another three months after that.

The present attack began on Thursday, March 16th, when she had abdominal pain, which has gradually increased. It was first felt near the umbilicus, but latterly has become localised in the left iliac fossa. During this time there has been complete constipation; no flatus has been passed, but a small quantity has come away after enemas, of which she has had three. The abdomen has been increasing in size, more especially during the last three or four days. Vomiting occurred first on Wednesday, the 22nd, and has been frequent. In the surgery, at 4.30, she vomited bilious fluid.

On admission, the patient was pale, with rather sunken eyes and a slightly anxious expression. The tongue was dry and covered with a whitish-brown fur. The abdomen was enormously distended, yet not exceedingly tense. For instance, the surface at any point could be depressed two or three inches, and be pushed, as it were, into the abdomen. The parietes were moving well with respiration. Distended intestinal coils became visible from time to time, but the peristalsis was not very active. Generally they appeared as two large coils about four inches wide, lying vertically one on either side of the umbilicus, while there was almost constantly a third hard vertical coil in the left flank, next to the left of the above two; but some movements were also observed in the right iliac fossa. No other tumour could be felt. There was not much tenderness, but a great deal of pain, so that she was constantly moaning and moving about uneasily in bed. Nothing could be felt per rectum; the rectum was empty and was not ballooned. The temperature was 98°, the pulse 80, the respirations 20. Another injection was given about 9 p.m. It caused a good deal of pain, but had no other result.

After consultation between us, it was decided to operate. The patient was put under an anæsthetic, and an incision was made of a length sufficient to determine the cause of the obstruction. A hugely distended coil presented in the wound, rendering it necessary to increase the length of the opening in the abdominal wall very considerably, in order to permit of its liberation. The loop proved to be the sigmoid

flexure twisted in such a manner as to obliterate its lumen. The distension of this piece of bowel was such that it measured eighteen inches in circumference and two and a half feet in length. The wall was very thin, translucent, and of a pink or yellow colour. The transverse colon was grey-green, two or three inches in diameter, with loculi still present. The small intestines were not distended. The entire volvulus was removed, a good-sized Murphy's button being used to establish continuity. A slight difficulty was experienced because of the great disproportion between the sectional areas of bowel and of the hypertrophy of the muscular wall of the proximal end. After the operation she was kept under morphia, and six ounces of peptonised milk were given as nutrient enemata. The bowels were opened twice on the 27th; the motions were loose and brown, and the second contained one or two small clots of blood. The temperature rose gradually to 100° and 101°, and the pulse quickened in proportion.

On April 2nd she had a little arrowroot by the mouth. The stitches were removed from the laparotomy wound on the 6th. The button was passed on April 25th, 31 days after the operation, and she went to a convalescent home on May 1st. The temperature, which had risen after the operation, continued above the normal for fully three weeks—between 100° and 102° the first week, between 99° and 101° the next five days, and gradually lower and lower the next nine days. The portion of bowel removed by operation and undistended was found to measure ten inches in circumference and two feet in length.

CASE II.—George B., æt. 68, was seen by Dr. Taylor with Dr. Bhedwar, of Southwark Park Road, on April 12th, 1902. He had been ill only a few days. It appears that he was well on the 7th, and went out with a friend to dinner, and he went to his business as usual on the morning of the 8th; but that afternoon he was seized with pain in the abdomen, and felt uncomfortable from distension. He took some opening medicine, but got no relief, and he saw Dr. Bhedwar on the evening of the 9th. The bowels, as a fact, had been opened last on the 6th, and since then there had been no motions in spite of enemas and

laxatives, and no passage of flatus in either direction. He was never sick at any time. The abdomen was very greatly distended and extremely tense, so that the surface could not be depressed more than a quarter or the third of an inch. It was resonant all over, and I did not see at the time any movements of peristalsis or any outline of intestines. The chest was much pressed upon by the distended bowel; his breathing was shallow and distressed, and the heart was pushed up, so that an impulse could be felt in the third space above the nipple. The patient was exhausted, the face dusky grey, the pulse small and feeble 105, the tongue dry.

It was arranged that he should go to Guy's Hospital. On his arrival he was very collapsed, with a temperature of 95° and a pulse of 116. Brandy was given internally, strychnine was injected subcutaneously, and a pint of saline solution was introduced by the rectum, but was not retained.

Mr. Lane made an incision parallel to, and one inch above, the left Poupart's ligament. He drew out a loop of colon, incised it, and let out a quantity of gas, but no fæces. The left half of the abdomen collapsed, but the right remained distended. A puncture was made to the right of the middle line, but without result. A Paul's tube was inserted into the incised gut, and the wound was only partially closed. The patient died before he could be got back to his bed.

The post-mortem examination was made by Dr. French. The sigmoid flexure, two feet in length, was twisted two half turns upon itself, the reverse way to a clock-hand, as one looked at it from the patient's feet. The part included in the volvulus had a perfectly glossy, smooth surface, without any trace of peritonitis. It was dark green in colour, and formed an immensely distended coil occupying the middle of the abdomen, and extending up to the ensiform cartilage, in front of all the other viscera. There was much congestion with hæmorrhage beneath the mucous membrane, the colour of which was deep crimson and in part black. There was a sharp line, above and below, where the normal mucous membrane changed into the deep crimson of the involved part. The circumference of the most distended coil



was thirteen inches. Above the volvulus the bowel was not greatly distended nor hypertrophied, nor were there any distension ulcers, even in the cæcum. The opening made at the operation was three and a half inches above the volvulus. The small intestines were normal, the stomach small and contracted. There were several small hæmorrhages in the tissue of the pancreas. The spleen was small, tough and dark, weighing only 61 grammes; the liver and kidneys were normal. With the exception of old adhesions of the lungs to the diaphragm and pericardium, and some pulmonary congestion and œdema, the thoracic organs were normal.

The points of interest to which we wish to call attention are the method of treatment in the first patient, and the course and duration of the symptoms of volvulus.

It was Mr. Lane's own suggestion that the twisted bowel should be entirely removed—an operation he had had in his mind for some time, and it certainly proved entirely successful and presented no great difficulties. The possibility of a recurrence of the volvulus, which has sometimes happened, and all the annoyances of an artificial anus, were thus obviated.

The wonderful usefulness of Dr. Murphy's mechanical contrivance was illustrated in a remarkable manner in this particular case, as any other method of effecting a junction between the extremities of the bowel would have been exceedingly difficult and tedious. With the button this was done in a very few minutes. The delay which would have resulted of necessity from the employment of any other method under the circumstances would have reduced very materially the chances of recovery of the patient.

Much care and time has been devoted to the attempt to discriminate between the different varieties of intestinal obstruction. In the present day this has often an academic rather than a practical interest, because the necessity of getting directly into touch with the obstructed bowel, whatever its nature or position, is universally recognised; and it must be confessed that in

neither of these cases was a diagnosis of volvulus made before the operation. Still a few remarks on the course and symptoms of volvulus may be permitted.

It is obvious that the results of an obstruction must depend much more upon its position in the length of the intestine, upon the rapidity of its occurrence, and upon its completeness, than upon the actual tissue-changes which are in operation; and so even with regard to a volvulus, which may affect the small intestine as well as the sigmoid flexure, one should not expect absolutely uniformity.

Volvulus most frequently involves the sigmoid flexure, and next most often the cæcum, and it is with regard to the former that most positive views have been expressed. From the writing of three authorities—Leichtenstern (in Ziemssen's *Encyclopædia*), the late Dr. Hilton Fagge, and Sir Frederick Treves—one gets the impression that the symptoms of volvulus are extremely severe and rapidly fatal. Treves (in *Allbutt's System of Medicine*) says out of twenty-one recorded cases only one patient lived until the sixth day; all the rest died within twenty-four or forty-eight hours. Fagge (*Practice of Medicine*, 8rd edition, 1891, vol. ii., p. 270), says:—"There is little delay in the occurrence of sickness, as in the other forms of obstruction of the large intestine; on the contrary, all the symptoms develop themselves with peculiar rapidity, the abdomen becomes quickly distended in the greatest possible degree, and death may occur within three or four days. Previous constipation is the rule. Volvulus is rare in women and children." Leichtenstern says death sometimes takes place within the first twenty-four hours, "on the average on the fourth day." The present cases suggest that those opinions have been formed on a limited number of cases, and that at any rate exceptions to such general rules may happily be found. Our first case was operated on upon the tenth day (nine days after the first symptom), and if we remember her condition rightly, we think she might well have held out another thirty-six or forty-eight hours. The man died during the operation on the fifth day, and his life was probably shortened only by a few hours, if at all.

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A parallel to the first case is found in one referred to by Dr. Benham and Mr. Silcock in a paper on Volvulus in the *Clinical Transactions*, vol. xxviii., 1895, p. 180, and first narrated in 1883 (*Lancet*, vol. ii.) by Mr. Clarke, of Glasgow. He says it "was in some respects remarkable, the obstruction having lasted about three weeks before operation seemed imperative, and was unattended throughout by vomiting." But curiously, even among the six cases in connection with which Dr. Hilton Fagge first formed his opinion of the rapid course of this lesion, we find three with durations of fourteen days, nine or ten days, sixteen or seventeen days, and one in which it is impossible to be sure, since the patient was vomiting for three or four months, and no data are given by which to fix the onset of the final period.

The second case no doubt falls more within the conception of volvulus as an acute and rapidly fatal disease. For the average of four days given by Leichtenstern, some cases as long as five, six or seven days must have occurred, and the case obviously falls within the limits of Leichtenstern's statement, and does not contradict that of Fagge, but is decidedly more promising than would have been expected from the records collected by Treves.

Another point of interest is the fact that vomiting may be of comparatively little importance. The male patient did not vomit at all; the female patient did not vomit for six days, and on the tenth day the vomited fluid was only bile-stained, and not stercoraceous. In Mr. Clarke's case just quoted there was no vomiting in an illness of three weeks. Of the six cases recorded by Dr. Fagge, in two the vomit was very little, and never faecal; in the third there is no statement of this symptom, and in a fourth, lasting nine or ten days, vomiting is only mentioned towards the end; in the fifth there was frequent vomiting, which was stercoraceous at the last; and in the sixth and quite anomalous case there were attacks of vomiting at intervals over six months, and the vomiting finally became continuous.

The symptoms in these two cases agree better with the account given by Messrs. Rose and Carless (*Manual of Surgery*, second edition, 1899, p. 972), who state that pain, vomiting and collapse

8     *Two Cases of Volvulus, one of which was Successfully  
Treated by Resection of the Twisted Bowel.*

are not so severe or marked as in other forms of strangulation, but that abdominal distension from excessive flatus and resulting dyspnoea or thoracic embarrassment are very distressing. This accumulation of gas is partly in the bowel above the lesion, but chiefly in the twisted loop itself. On the other hand, these cases form an exception to their statement that plastic peritonitis soon fixes the coil, for in neither case was there sufficient peritoneal inflammation about the neck of the volvulus to prevent easy manipulation.

We do not wish to draw hasty conclusions from a few instances but only offer them as a contribution to the subject. Indeed, a consideration of all the cases seems to us to show that generalisation from a few instances can rarely give correct views, and that neither are the symptoms of particular pathological lesions so uniform, nor are the distinctions between the symptoms of different lesions so constant, as we should, for our convenience, like to believe.

# ON THE PATHOLOGY OF ACUTE RHEUMATOID ARTHRITIS.

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It has occurred to several writers that the cases which are often grouped together as one disease under the names Arthritis Deformans, Osteo-Arthritis, or Rheumatoid Arthritis, differ so much among themselves that it will ultimately be shown that these terms, as now used, include two or more separate diseases. There have lately been under my care in Mary ward two excellent examples of that comparatively rare condition, the acute form, and I have been accustomed at the bedside<sup>1</sup> to point out how strikingly it differs from, on the one hand, rheumatic fever, and on the other from the other forms of disease comprehended under these three terms, arthritis deformans, osteo-arthritis, and rheumatoid arthritis. When these two patients were in the hospital I had not read the articles of Dr. A. E. Garrod<sup>2</sup> and Dr. G. A. Bannatyne,<sup>3</sup> but I now find that they too are much struck by the differences between these acute cases and other patients said to be suffering from rheumatoid arthritis. I think, from carefully reading their articles, I may say that Dr. Bannatyne, Dr. Garrod and myself have independently arrived

<sup>1</sup> Clinical Journal, Dec. 12, 1900, p. 113, and May 29, 1901, p. 84

<sup>2</sup> Brit. Med. Journ., vol. 2, 1901.

<sup>3</sup> Practitioner, May, 1900, p. 530.

at the conclusion that this acute form is a separate disease, which should be distinguished from the others, and should not, as it is in many text-books, be described with the chronic forms. As I shall show directly, probably Dr. Bannatyne has included in his description some of those rare cases in which permanent damage to joints is left after rheumatic fever, but to him belongs the credit of having insisted two years ago on the separation of this acute form, and therefore I should not now have written this paper had it not been that no detailed account of a post-mortem examination made upon a patient suffering from this disease has as yet been published. Dr. A. E. Garrod, when he opened the discussion on "Chronic diseases of joints commonly included in the terms chronic rheumatism, osteo-arthritis and rheumatic gout," at the Cheltenham Meeting of the British Medical Association in 1901, said, speaking of the acute form, "Our knowledge of the morbid anatomy of this disease is very scanty. It has so little fatal tendency in its earlier stages that opportunities of examining such joints post-mortem are very rare. In our London hospitals such opportunities are practically wanting, and I believe that we could have no more valuable contribution to this discussion than a careful description of the post-mortem appearances of an early stage of this kind of rheumatoid arthritis." No one joining in the discussion supplied the description Dr. Garrod wanted, and hence the publication of the following account of a post-mortem on a patient who had the disease. I have added the description of the clinical features of a second patient suffering from this disease, and who is still alive, not only to illustrate the symptoms, but because the radiographs from both the cases suggest that future observations may show that the radiographic appearances of this disease are peculiar. These will be considered at the end of this paper.

Before describing the individual patients it will be well to attempt to classify the cases comprehended under the terms arthritis deformans, osteo-arthritis, and rheumatoid arthritis. Dr. Bannatyne believes that this group contains three distinct diseases, and Dr. Garrod, too, makes three, and it certainly appears to me that in this these authors are correct. There is

*On the Pathology of Acute Rheumatoid Arthritis.*



FIG. 1.





the disease usually called osteo-arthritis, often confined to the hip-joint and commonly seen in elderly men. Secondly there is the disease frequently met with in women in middle life in whom the trouble commonly begins in the terminal joints of the fingers and the carpo-metacarpal joints of the thumb. The deformity is considerable and is ultimately largely due to bony out growth; in a large number of cases the trouble gradually spreads until many joints are implicated and the woman may become a helpless cripple, and although in one sense she is severely ill yet the disease is very slow, the symptoms are not acute and there is no pyrexia. Thirdly, we much less often meet with cases occurring for the most part in women whose age is usually near to twenty, in whom the disease begins, or is at any rate soon most prominent in the proximal phalangeal joints and is quite early strikingly seen in the wrists, it is markedly symmetrical in the two hands, the swelling is considerable and extends beyond the joints so that there is a well marked fusiform swelling about the affected interphalangeal joints and a general swelling about the wrist and other joints; sometimes a creaking may be felt, due apparently to thickening of the synovial membrane. (See Figs. 1, 2 and 4). In the course of a very few weeks most of the joints in the body are affected, so that the woman is soon bedridden, unable to move fingers, thumbs, wrists, elbows, shoulders, and the corresponding joints of the lower extremity. The joints of the spine are often affected so that turning in bed is difficult and painful; the temporo-maxillary joint is frequently implicated and mastication is consequently difficult.

Wherever the disposition of surrounding muscles makes it easy to investigate the joints, swelling about them is obvious and the patient usually complains of pain in them. Muscular wasting is so very rapid that it is clearly an extreme degree of genuine arthritic atrophy not dependent on disuse; the wasting is especially seen in the hand and forearm, but in a bad case it is almost universal. The temperature is often raised daily, mostly in the evening, to a point usually between 100° and 102° F. (see chart of case 2). The hands and feet are often covered with sweat, the patient is pale and the pulse is increased in frequency

out of proportion to and apart from pyrexia (see chart of case 2) although neither from the history nor from examination of the heart is there any evidence to connect the disease with rheumatic fever.

After three or four weeks the temperature slowly regains its normal point (see chart of case 2), the rate of the pulse diminishes, but more slowly than the temperature. The colour partly returns, the sweating of the hands and feet lessens, the pain passes away, but much immobility and some swelling around the joints remain, and the patient is liable to fresh attacks which exactly resemble the first and result in fresh swelling of and around the already fixed joints. After the acute stage has subsided the sufferer is by no means recovered, for the swelling of the synovial membrane and soft tissues around the joint leads to great immobility of them and much of the muscular atrophy remains; the patient is hence often a chronic invalid. In this respect, as well as in others, the disease stands in striking contrast to nearly all cases of rheumatic fever. These points will all be illustrated in the cases I am about to relate.

Careful manual examination during life even in cases which have lasted many years fails to reveal any lipping or bony outgrowths in connection with the joint. The deformity appears to be entirely due to effusion into the joint and swelling of the tissues around. Radiographic examination entirely confirms this view.

Mr. Shenton has kindly taken for me many radiographic pictures of the hands, wrists, elbows, shoulders, knees and ankles, and often the same joint has been examined at intervals of several months. Some of these radiograms are published at the end of this paper, and it will be seen that there is not the slightest trace of bony outgrowth, even in a case which had lasted eight years. The description of the post-mortem examination on the first case amply confirms this view, for no bony outgrowths or eburnation were present (see p. 18), and leads us to believe that the disease is essentially one of the synovial membrane and the soft tissues around the joint, the very slight change in the cartilage and bone being apparently secondary

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FIG. 2.



to that of the synovial membrane. Probably the disease is of microbic origin, a diplococcus was found in my case (see p. 21), and other observers have found micro-organisms, but the specific micro-organism has not yet been certainly isolated.

No treatment is at present known that will cut short the acute stage, which lasts about four weeks; towards the end of that time the temperature has become normal, the pulse is slower and the pain has gone, but much swelling and great immobility of the joints remain. Much good can now be done by passive movements and massage, for not only is the revival of the atrophied muscles hastened but the permanent fixation of the joints, which the patient should be encouraged to move voluntarily, is to some extent prevented. Probably, too, heat locally applied is helpful, but as the number of joints affected is so great and the wasting of muscles is so marked, striking deformities and contractions are especially liable to be left behind after an acute attack. The points will be illustrated by the two cases presently to be related.

With regard to a name, that of acute rheumatoid arthritis, suggested by Dr. A. E. Garrod, is probably the best, for the disease is acute at first and even later on acute attacks are liable to occur, the absence of marked changes in the cartilage and bone makes it resemble rheumatic fever and arthritis certainly is present.

The above description was written before I read Dr. Bannatyne's, and probably anyone reading the two will see that we are each describing the same disease. The points of difference are that I am not so struck with the majority of cases "following on some infective process as tonsillitis, influenza, rheumatism, &c." Tonsillitis, influenza and other slight infective diseases are so common that it would be an extremely difficult matter to show that there was any relationship between them and acute rheumatoid arthritis. It may be that there is, but extensive statistics are necessary to prove the point. If by rheumatism he means rheumatic fever I should join issue with him. As far as my experience goes, sufferers from acute rheumatoid arthritis have not usually had rheumatic fever nor any of the diseases

commonly associated with it, *e.g.*, endocarditis, pericarditis, chorea or erythema. Further, the proximal finger-joints, the temporo-maxillary joints and the spinal joints are commonly affected in acute rheumatoid arthritis but not in rheumatic fever. That rheumatic fever may occasionally be followed by permanent changes in the joints is certain, but probably such changes have nothing to do with the disease in question, which, by-the-by, is not benefited by salicylic acid. Neither do the cases I have seen show "numerous nerve symptoms," nor during their "course such complications as pneumonia, pleurisy, pericarditis, endocarditis, etc." I cannot help thinking from his mentioning these that Dr. Bannatyne has included among his cases some of those rare cases in which permanent joint trouble remains after rheumatic fever. He does not state what is the order in which the joints are most commonly implicated, but Dr. Garrod agrees that it is that just mentioned, but Dr. Bannatyne speaking of the acute stage points out what is very important, namely, that there are no bony outgrowths or lipping at the edges of the joints. He does, however, say that when the disease has lasted some time "Atrophy is marked especially from disuse; and cartilaginous and even bony outgrowths are seen round the margin of the joints as well as in the tendons, bursæ, etc." The patient whose case is about to be related had had the disease eight years, and yet at the autopsy not a trace of cartilaginous or bony outgrowth, either round the joints or near the tendons or bursæ could be seen, and this case and others observed during life lead me to think it will be found that the absence of lipping and outgrowths is usually striking, not only in the acute but also in the later stages of the malady. Further, the arthritic muscular atrophy is so manifest and so extreme that in severe cases it quite overshadows atrophy from disuse. I have not been struck, as has Dr. Bannatyne, with the enlargement of the glands proximal to the affected joints, nor with many trophic and vasomotor changes, but in the main the cases that have come under my notice conform with his account of this disease. Any differences are probably to be ascribed to his having included some cases of joint trouble following rheumatic fever, and



possibly some of the more severe cases of the osteo-arthritis usually seen in older women who often show Heberden's nodes very strikingly, but who are really in all probability afflicted with a different disease from acute rheumatoid arthritis. He does not describe the post-mortem appearances of an individual case but gives a general description which tallies very closely with the condition about to be recorded.

The age of incidence, the symptoms, the definite course of the acute attacks and the pathological findings, all point to this acute rheumatoid arthritis being a distinct disease. As already mentioned, although permanent joint deformity may occasionally follow rheumatic fever, acute rheumatoid arthritis has its own preferential joints, *e.g.*, the proximal interphalangeal, wrists, temporo-maxillary and spine, and of these the wrist is the only one commonly affected in rheumatic fever; patients with acute rheumatoid arthritis do not give a history of previous rheumatic fever, of chorea, of rheumatic nodules, nor do they show any special liability to endocarditis or pericarditis, nor are they benefited by salicylic acid. The above reasons tell equally against regarding acute rheumatoid arthritis as merely a variety of rheumatic fever.

The distinction from the chronic osteo-arthritis seen chiefly in middle aged women seems equally marked, for these patients are older. Their disease is not acute, pyrexia is rare and transient, it is the terminal phalangeal joints that are particularly and commonly affected as we see in the presence of Heberden's nodes, and radiographic and post-mortem evidence shows that bony outgrowths, eburnation and bony grating are common. The progress is very slow, it is usually several years before as many joints are implicated as may in acute rheumatoid arthritis be affected in a fortnight, and we do not in this chronic osteo-arthritis see the characteristic spindle-shaped swelling about the proximal interphalangeal joints, nor is the arthritic atrophy so striking.

Probably no one would maintain that the senile osteo-arthritis, gonorrhœal arthritis, traumatic arthritis, septic arthritis, tubercular arthritis, hæmophilic arthritis and the nervous arthritis of

tabes or syringomyelia have anything to do with acute rheumatoid arthritis, but whether this disease is in any way connected with the form of chronic joint disease of children which was described by Still<sup>4</sup> is uncertain. It is true that in his case the enlargement of the joints felt and looked more like "a general thickening of the tissues around the joint than a bony enlargement and is correspondingly smooth and fusiform with none of the bony irregularity of the rheumatoid arthritis of adults. The absence of osteophytic growth and of anything like bone lipping, even after years have elapsed since the onset, is striking." But, on the other hand, the enlargement of the lymphatic glands, the enlargement of the spleen and the presence of adherent pericardium in Still's cases seem to separate the two conditions.

I will now give a description of the two cases, and will finally describe the appearances seen by the Röntgen rays in both.

CASE 1.—Kate B., æt. 20, was admitted into Guy's Hospital on July 31st, 1901, under the care of Dr. Hale White; she was unmarried and a servant. She entered service at the age of fifteen and has lived in London all her life. The family history is unimportant.

The patient is said to have had pleurisy and congestion of the lungs at the age of eight. She had an attack similar to the present in September, 1894. Her hands were the first to become swollen, but later her knees and ankles were affected. This attack lasted on and off for about twelve months and during it her wrists became fixed and she has been unable to bend them since; part of this time she was an out-patient at the Waterloo Road Hospital. After this attack she was free until February, 1896, when she suffered in the same way again, the knees being first affected, but other joints were soon swollen and painful. She was an out-patient at St. Thomas's Hospital for seven months and then went to the Bath Hospital for two months. This attack was about the same in severity as the first; it lasted eighteen months. From October, 1897, she was free until

<sup>4</sup> *Medico-Chirurgical Transactions*, Vol. 80, p. 47.

Christmas, 1899, when she had an illness that was called rheumatic fever, but it was apparently the same as the other attacks and there is no history that the patient has had chorea or any other of the rheumatic diseases. She had another attack in October, 1900. The present illness began at the end of February, 1901. It started with swelling in the knees, and she was unable to walk. The swelling next appeared in her hands, and then in her feet and shoulders, and lastly she could not move her neck or her hips. She has been in bed ever since this attack started, has lost a great deal of flesh, and has been getting paler.

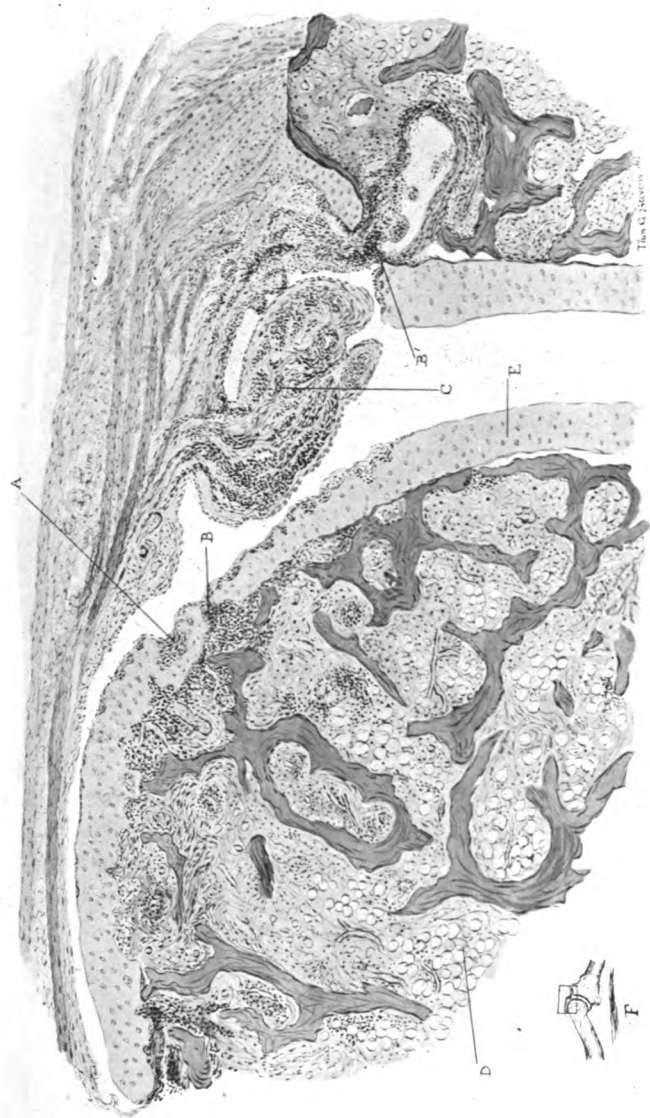
*On admission.*—She was very pale, especially about the lips. Her pulse was 116 and her temperature 99·6° F. There was great atrophy of all the muscles of her limbs and it was especially marked in the forearms and hands. All the joints of the hands, both wrists, elbows and shoulders, both hips, knees and ankles, and as far as could be made out the joints of the feet, were fixed, most of them completely. The implication of the temporomaxillary joint was shown by the fact that she could open the mouth but a little way and difficulty of certain movements suggested that the joints of the spine were affected. There was swelling about many of the affected joints. It was especially marked around the proximal interphalangeal joints (which showed the characteristic spindle-shaped swelling), around the wrists, knees and ankles. Figs. 1 and 2, from photographs kindly taken by Mr. H. Watts, illustrate these points admirably, and the contrast between the slight swelling of the terminal and the great swelling of the proximal phalangeal joints is especially marked. Nowhere was there the slightest evidence of bony outgrowths and the swelling certainly appeared to be as much a general thickening of the tissues around the joints as an excess of fluid in them. In the knees a creaking sensation could be felt, such as might be due to thickening of the synovial membrane. Over some of the joints the skin was slightly reddened. There was some pain, but little tenderness.

There was nothing abnormal to be made out in the heart, lungs or abdomen; the hands and feet sweated considerably. During August and September the condition slowly became less

acute, the temperature after some weeks being nearly normal, the pulse became less frequent, the swelling about the joints diminished ; she regained a little muscle and the sweating of the palms and soles was less striking. On October 10th the temperature was 104° and she had a rash which looked like that of measles. This soon disappeared and she gradually sank into a typhoid condition and died on October 14th. She gave a Widal reaction with a 5 per cent. dilution and the case was regarded as one of typhoid fever with a premonitory rash. This view was supported by finding at the post-mortem a congested Peyer's patch in the ileum and an enlarged spleen ; but cultivations taken from the spleen did not show any typhoid bacilli. The cause of death cannot be said to have been certainly determined.

Many joints were examined at the post-mortem examination by Dr. Fawcett. There was some thickening of the tissues on either side of the phalangeal joints of the fingers and of those on either side of the knees. It was especially marked around the proximal interphalangeal joints. The following joints were opened : the right knee ; here the synovial membrane was thickened and of a pinkish red colour ; on its inner surface were a few shreds of lymph ; there was a very slight erosion of the cartilage on the external condyle but the rest of the cartilage was healthy. There was an excess of synovial fluid in the joint but no outgrowths from the bones or the edges of the cartilages. The whole condition suggested a chronic inflammation but with the affection of the joint itself almost limited to the synovial membrane. The right ankle-joint was opened, also the temporo-maxillary and one of the finger-joints ; but in these, except perhaps for a little excess of synovial fluid, there was absolutely nothing abnormal to be seen by the naked eye. The whole disease was apparently not an affection of cartilages or bones but rather an affection of synovial membrane with swelling of the tissues outside the joints. Nowhere was there any sign of bony outgrowth.

One of the most affected proximal interphalangeal joints together with parts of the shafts of each of the adjacent





phalanges and the coverings of the joint, was reserved for histological examination. Sections of it were kindly prepared by Dr. T. G. Stevens. The whole specimen was decalcified in 70 per cent. alcohol, with 3 per cent. nitric acid, the process taking about six weeks. The specimen was then embedded and sections were cut with a freezing microtome. They were stained with hæmatoxylin and faintly counter-stained with fuchsine "S" and orange "G."

The greatest changes are seen at the dorsal part of the periphery of the joint. Here the synovial membrane is thickened by the formation of new fibrous tissue, and a thickened projecting fringe of synovial membrane (C.—Fig. 3) is especially evident. In this evidences of both chronic and recent inflammation can be seen. The synovial membrane on the cartilage opposite to this thickened fringe is chronically inflamed (A and B.—Fig. 8) and thickened, new fibrous tissue being formed. The cartilage underlying this thickened membrane is thinned and eroded, and in one place the erosion has perforated the cartilage (B.—Fig. 3). The bone in the neighbourhood is the seat of chronic inflammatory changes; the cancellous tissue is more open than usual. Also, there are foci of more recent inflammation in the bone, as shown by small-celled exudations here and there. In these parts of the bone there is a comparative absence of fat cells compared with the deeper parts of the bone. But a reference to the figure will show that all these changes in the bone and cartilage are slight in comparison with those in the synovial membrane, and it is very striking to observe how they are opposite to the markedly inflamed synovial fringe, as though set up by direct contact with it. The extremity of the cartilage of the other phalanx is perforated in the neighbourhood of the thickened fringe (B<sup>1</sup>.—Fig. 3), and the bone subjacent to the perforation shows the same changes as those already described. Here also it appears as though the pressure of the synovial fringe had determined the perforation of cartilage, and it is especially to be noticed that over both phalanges the cartilage is perfectly normal, except for the perforations and erosions just mentioned. There are similar but much slighter changes in the palmar edge

of the joint; all the intervening part is quite healthy. There is not the slightest evidence of proliferation of bone or cartilage, and there is every appearance that the histological changes, which are slight considering the many years the disease has lasted, began in and are chiefly confined to the synovial membrane. The erosions and perforations of cartilage were so trivial as to be invisible to the naked eye. As will be mentioned directly, however, when considering the Röntgen-ray appearances, there is probably some chemical changes in the ends of the bones.

Outside the joint there is considerable increase of fibrous tissue producing thickening of the ligaments.

It will be observed that the histological examination of this joint does not support Bannatyne's statement "that in the acute variety the essential characteristics are an acutely destructive polyarticular and general disease," for there was but little destruction of the joint, although the disease had lasted many years.

Sections of one of the synovial fringes of the knee showed exactly the same changes. The fringe was much thickened and it contained a quantity of new fibrous tissue, together with a number of small cells and many new formed vessels.

Specimens of the following were taken with all proper precautions and sent to the Hospital bacteriological laboratory: (1) blood from right ventricle, (2) fluid from the right knee, (3) mesenteric gland, (4) spleen, (5) scraping from the synovial membrane of the left knee. Specimens 1, 2 and 4 were sterile. From the mesenteric gland a small non-mobile bacillus was obtained which stained by Gram's method but could not be identified. From the synovial membrane of the left knee a coccus was obtained which apparently was not Paine and Poynton's, nor was it like any of the more usual cocci. Neither Dr. Washbourn nor Mr. Pakes could identify it. Intravenous and subcutaneous injection of rabbits caused no symptoms. The following are the full reports of these micro-organisms kindly supplied by the bacteriological laboratory:—



P.M. 280.—MESENTERIC GLAND.

*Description*.—Rather small bacillus mostly in pairs and short chains.

*Spores*.—Not observed.

*Staining reactions*.—Carbolic methylene blue = fair. Gram = well.

*Broth*.—Fair growth, no gas, no indol.

*Gelatin stab*.—Not very abundant growth, whitish, not spreading on surface, no liquefaction, no gas.

*Gelatin streak*.—Scanty whitish, not spreading, semi-transparent confluent growth.

*Agar*.—Small, almost transparent, raised, moist, whitish colonies, like those of streptococci.

*Blood-serum*.—Very minute greyish colonies in two days.

*Potato*.—No visible growth.

*Litmus milk*.—Turned distinctly acid and clotted in four days.

*Peptone water*.—Very feeble growth.

*Litmus broth*.—Containing :

*Dextrose*.—Slight growth, acidity, no gas.

*Lactose*.— Do. do.

*Saccharose*.— Do. do.

*Glycerine*.—Neutral, no gas.

*Nitrate*.—Slight growth, no reduction to nitrites.

*Lead*.—Very feeble growth, no H<sub>2</sub>S produced.

*Anærobiosis*.—Poor anærobe, giving slight turbidity in broth (glucose formate).

*Pathogenicity*.—Mouse injected subcutaneously, not affected at all. Guinea-pig injected subcutaneously, died in nineteen days, no lesions found, no growth on media planted from organs. Animal probably died from other cause.

P.M. 280.—SYNOVIAL MEMBRANE OF LEFT KNEE.

*Description*.—Coccus, chiefly single, some pairs *but no chains*.

*Staining reactions*.—Well with carbolic methylene blue and by Gram's method.

*Broth*.—Very fine granular precipitate hanging to side of tube, no indol.

*Gelatin stab.*—Small, yellowish discrete colonies along needle track, no gas, no liquefaction.

*Gelatin streak.*—Small, white discrete colonies, somewhat raised, no liquefaction.

*Agar.*—Small, round, whitish raised colonies.

*Blood-serum.*—Discrete, moist, raised greyish white colonies.

*Potato.*—Not visible.

*Litmus milk.*—Turned faintly acid, not clotted.

*Litmus broth.*—Containing :

*Dextrose.*—Good growth, acid, no gas.

*Lactose.*—Slight growth, neutral.

*Nitrate.*—Good growth, no nitrites produced.

*Lead.*—Slight growth, no  $H_2S$ .

*Anærobiosis.*—Fairly good floccular growth in glucose formate broth.

*Pathogenicity.*—Mouse inoculated subcutaneously recovered. Guinea-pig inoculated subcutaneously died in nineteen days, on the same day and probably same cause as the other. Rabbits injected intravenously, not affected.

These observations have been recorded in this detail because the bacteriology of the disease is still quite uncertain, and therefore they may be useful for future workers. Bannatyne, working with Wohlmann and Blackall, has described a small bacillus, which as it stains at the two ends, looks like a diplococcus. This, he considers, is probably the same as that described by Chaffard and Ramond. Reference is often made to a micro-organism described by von Dungern and Schneider,<sup>5</sup> but the reader will find that their patient had had rheumatic fever and that endocarditis was found at the post-mortem, so the observations of these authors are of more interest in connection with rheumatic fever than in connection with acute rheumatoid arthritis.

This case shows that the pathological changes in acute rheumatoid arthritis consist of a chronic inflammation of the synovial membrane together with a thickening of the tissues

outside the joint. The pitting of the cartilage is slight and secondary to the changes in the synovial membrane. The cartilage itself is healthy and the affection of the bone is slight and due to perforation of the cartilage by the thickened synovial membrane. There are no bony or cartilagenous outgrowths.

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CASE 2.—Isabella S., a waitress, was admitted into Mary ward under Dr. Hale White on September 15th, 1900. There was no history of rheumatic fever either in the patient's family or in herself. Six months ago she first had pain and swelling of the feet and ankles and soon after in the left hand, wrist and elbow. Two months later the joints of the right upper extremity were affected in a similar way.

*On admission.*—There is no joint in the body the movement of which is certainly perfect, and in most movement is impossible so that she lies in bed almost helpless. All the proximal interphalangeal and the metacarpo-phalangeal joints and both wrists and knees are much swollen. There is also some swelling about the elbows, ankles and small joints of the feet. She cannot move her hips, or shoulders, movement of the spine is restricted and difficult, and the mouth can only be opened a little way on account of affection of the temporo-maxillary joint. It is clear that the swelling is due more to affection of the tissues round the joints than to excess of fluid in them. This is strikingly evident in the wrists and knees but especially in the proximal interphalangeal joints, where there is a fusiform swelling around the joint which extends some distance along each of the adjacent digits. The joints are not hot nor red, and considering the pain, the tenderness is very slight. The muscles are markedly wasted. She is very anæmic, the hands and feet perspire profusely. For the first four weeks of her stay in the hospital her temperature was raised every day to about  $101^{\circ}$  (see Chart, Fig. 5) and did not fall quite to normal in the morning. The pulse was much more rapid than the temperature would explain. Slowly the temperature fell to normal in between three and four weeks, the pulse became less rapid, the sweating diminished, the swelling around the joints became less and the pain disappeared. When

the acute symptoms had decreased sufficiently she was massaged all over daily and passive movement of the joints was practised. Under this treatment the muscle became less wasted and considerable mobility of joints was restored, but when she left the hospital on December 20th, 1900, movement of joints was still much restricted and there was considerable wasting; she still sweated a little on the palms and soles; her colour was pale but less so than on admission; her pulse was only a little increased in frequency; her temperature was healthy, and she was on the whole much better than on admission, suffering chiefly from the inability to move perfectly any of her joints.

She was re-admitted into Mary on February 25th, 1902, at my request, so that we might see how she had progressed. She was now not anæmic, the sweating of the soles and palms was slight, her temperature was normal, her pulse about 80, the wasting was less and she said she felt much better. She could open the jaw almost as wide as healthy people, and she could bend her hips well so that she could sit up in bed, but movements of the spine were by no means free. The shoulders, elbows and wrists were quite fixed and the fingers almost so. She could, however, walk if held by someone on either side, but not with sticks because she could not grasp them. There was considerable swelling of the tissues around the knee, less round the wrists and proximal interphalangeal joints, which still showed very well the fusiform form of swelling. (See Fig. 4). There was no appreciable swelling around the other joints. There was some creaking in the knee just like that given by thickened synovial membrane. There was no evidence of the formation of new bone, either from feeling the joints or by the aid of the Röntgen rays. Contrasting her condition with that on her discharge on December 20th, 1900, it was difficult not to consider that had she when at home had the advantage of frequent massage and manipulation of the joints, much of the fixation of joints would have been avoided and the muscles would have been less wasted. This view was supported by the fact that the temporo-maxillary joint which she was frequently using had almost regained its normal range of movement,



FIG. 4.



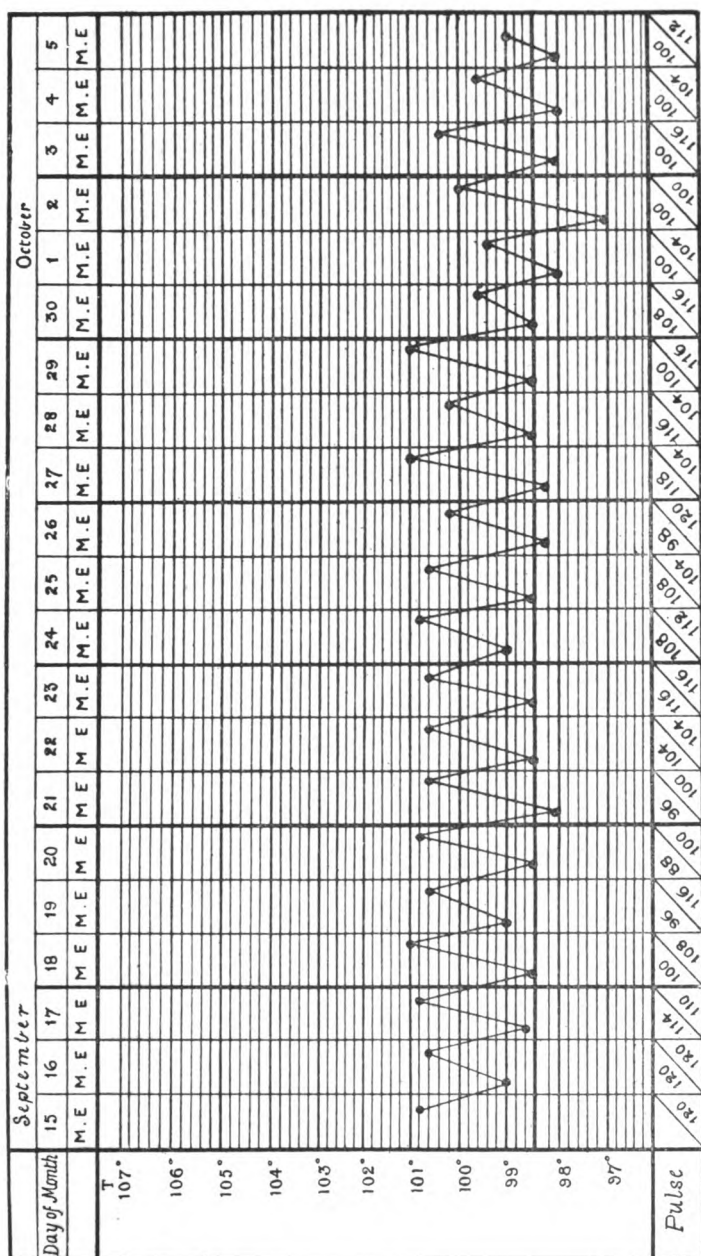


FIG. 5.

We will now pass on to consider the Röntgen ray appearances in these two cases. Fig. 6 shows the hand from our first case (Kate B), and Fig. 7 the hand from our second case (Isabella S.) on her first admission. The thing that strikes us on examining these pictures is the unusually transparent appearance of the ends of the bones. In both hands it is best seen either side of the metacarpo-phalangeal joints, but it is also quite evident at the ends of the phalanges. In the elbow, too, it was noticeable, especially in the olecranon, and other radiographs from these patients showed it in the ends of the bones forming the shoulders, ankles and knees. I have looked at many radiographs of healthy joints and also at those taken from patients suffering from chronic osteo-arthritis, and although the ends of shafts are often more transparent than the middle, comparison with my two cases does suggest that possibly in future an exceptional transparency to the Röntgen rays of the ends of the bones will be regarded as peculiar to acute rheumatoid arthritis when this disease is contrasted with those having a very similar name. Two cases are too few for a generalization, but this suggestion gains force from the fact that in looking through a number of radiographs of hands I came across one in which transparency of the ends of the bones was striking. The radiograph was labelled "chronic osteo-arthritis" and I was unable to trace the case, but the radiograph showed no trace of bony outgrowth or lipping, which in itself was suggestive that the disease from which the patient was suffering was not chronic osteo-arthritis but acute rheumatoid arthritis, and this view was supported by the fact that there was extreme flexion of the phalanges at the proximal interphalangeal joints. Now, as in acute rheumatoid arthritis, these joints are most strikingly affected and the muscular atrophy is extreme, the secondary flexion due to contracture is especially likely to occur at the proximal interphalangeal joints. Again, the suggestion here made that this exceptional transparency of bone may some day be found to be one of the distinctions of acute rheumatoid arthritis from other diseases having a similar name, is indirectly supported by



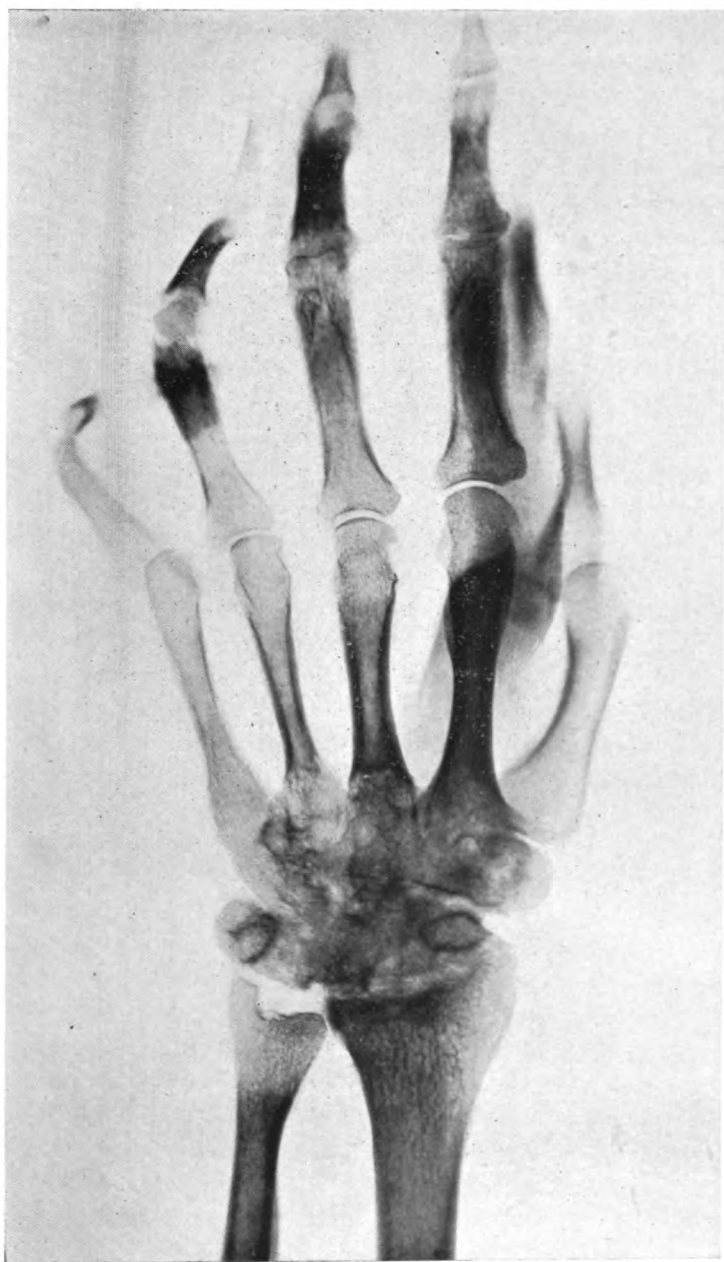


FIG. 6.





FIG. 7



Walsh,<sup>6</sup> who says, "The rheumatoid bones look more fragile; in point of fact they are atrophied, a condition which renders them more penetrable to the rays. Indeed, from their appearance it may be pretty safely inferred that they are deficient in bone salts." Later on he speaks of their greater transparency. The radiograph he publishes to illustrate the point is taken from a lady, aged 31, who had had the disease since the age of 13, and nearly every joint in her body was affected. But it is very likely that this patient was really suffering from acute rheumatoid arthritis, for I have pointed out that this disease usually occurs in young women, that its resulting deformities continue for many years and may affect every joint in the body. Whether this transparency is greater in acute rheumatoid arthritis than in other diseases of joints, such as tubercle, osteomalacia, and cancer, in which Walsh says it may occur, is a point well worthy of further investigation.

The radiographs from Isabella S., were taken in October, 1900, by Mr. Shenton, when she was under my care. The unusual transparency at once struck all of us who saw the pictures, and Mr. Shenton and I both referred very shortly to the point in the *Clinical Journal*<sup>7</sup> and since then we have observed the same appearances in the radiographs taken from Kate B. (Fig. 5), but we were not the first to observe it, for the first edition of Walsh's book was published in 1897. This transparency of bone is not transitory, for the patient who was in the hospital twice showed it on each occasion.

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#### DESCRIPTION OF FIGURES.

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Fig. 1.—Shows the deformity of the wrists, finger-joints, knees and ankles, in the case of Kate B.

Fig. 2.—Shows the swelling of the wrists, metacarpo-phalangeal and phalangeal joints of Kate B. It illustrates the extreme change about the proximal phalangeal joints, causing the fusiform swelling about them.

<sup>6</sup> Walsh "The Röntgen Rays in Medical Work," 3rd edition, p. 161.

<sup>7</sup> *Clinical Journal*, May 29th, 1901, pp. 84 and 85.

Fig. 3.—A section of part of one of the proximal interphalangeal joints. The part included in the rectangle in the small figure F shows the part shown in the larger picture which represents a magnification of 45 diameters. The bone to the right is first phalanx, that to the left is second phalanx.

- A. Eroded cartilage with slightly thickened synovial membrane on it.
- B.B<sup>1</sup>. Perforation of cartilage by synovial membrane.
- c. Thickened fringe of synovial membrane. This contains an increase of fibrous tissue and also evidence of recent acute change.
- D. Normal medulla of bone.
- E. Normal cartilage of joint.

Fig. 4.—Isabella S.'s hand on her second admission. The figure shows very well the swelling about the wrists and fingers especially the proximal phalangeal joints. The photograph was taken sixteen months after the subsidence of an acute attack.

Fig. 5.—Chart showing the pulse and temperature during the acute attack from which Isabella S. suffered during her first stay in the hospital.

Fig. 6.—Shows the transparency of the bones of the hand in the case of Kate B. It is especially well shown at the ends of the bones. It will be observed that she is supporting the hand with one finger of the other.

Fig. 7.—Shows the transparency of the ends of phalanges and metacarpal bones in Isabella S. The swelling of the soft tissues around the proximal interphalangeal joints is very evident.

# THE CONDITION OF THE BLOOD IN PNEUMONIA TOGETHER WITH SOME RECORDS OF BLOOD EXAMINATIONS DURING THE HEALING OF WOUNDS.

(THESIS FOR THE M.D. CAMBRIDGE.)

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By H. A. GAITSKELL, M.D.

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## PART I.

### CONDITION OF THE BLOOD IN PNEUMONIA.

IN the following account of the blood in pneumonia I have left untouched the question of bacteriology and of coagulability of the blood and have chiefly devoted attention to the corpuscular elements. A free use has been made of all available literature on the subject, and a somewhat long, but I hope complete, summary of important facts bearing on the matter is appended. A detailed account is given of the principal points observed in technique, as undoubtedly the value of any blood examination is enhanced, and the work more accurately gauged if such a practice is adopted. The reports of cases, perhaps, take up an excessive amount of space, but, particular attention has been paid to the recounting of physical signs in order that their relation to alterations in the corpuscular elements may be more readily appreciated when comparison is made with the charts and tables.

## LITERATURE.

*The red cells.*—Cabot (Guide to Clin. Exam. of Blood, 1900) says that during the fever the red cells are approximately normal (unless increased by cyanosis); but after the crisis there is often slight anæmia, due partly to blood destruction, evidenced by the frequent appearance of hydrobilirubin in the urine.

Ewing (Clin. Path. of Blood, 1901) agrees with this statement, but says, "This result can be referred only to the concentrating effects of fever exudation, and local phenomena, and is to be seen in other infectious fevers."

In spite of these complicating factors, Sorenson, Boechman, Halla, Tumas, Sadler, and others have shown that the red cells usually show a slight but steady decline during the course of the fever. The decrease of red cells often occurs suddenly with the crisis, Tumas (Deut. Archiv. Klin. Med. Bd. 29) reporting one case with a fall of 600,000 on the day of defervescence. Most observers agree that the grade of anæmia established is usually not very marked.

Türk (Klin. Blunt. Wien., 1898) found nucleated red cells in seven out of eighteen cases examined. These were usually normoblasts, occasionally megaloblasts. Maragliano has noticed degenerative changes in the red cells, but no other observer refers to this.

*Hæmoglobin.*—Most writers are of opinion that the hæmoglobin is more reduced in proportion than the red cells, and Ewing says that in the absence of complications it seldom falls below 65 per cent.

*Leucocytes.*—It is in the leucocytes that well marked changes are to be observed in the blood in cases of pneumonia; consequently there is an enormous amount of literature to be found on the subject. Only the most important of these papers are referred to here, but many authors are quoted without references being given; partly because they are cited by other authors, and partly because too much space would be occupied by so doing.

That the leucocytes in the blood of persons suffering from pneumonia are increased in number was first observed by Piorry, and confirmed by Virchow, Nasse, and other early writers.



Halla, in 1883 (*Prag-Heilk*, 4, Bd. S, 198, 1883), was the first to recognise cases in which leucocytosis was absent. He also noticed in those cases ending in recovery that there is no correspondence between the curves of temperature and the curves of leucocytes. He encountered three cases in which leucocytosis was absent, all of which ended fatally.

Hayem and Gilbert (*Arch. Gen. d. Med.*, p. 257, 1884), recorded other cases where leucocytosis was absent, all of which were fatal.

Tumas (*Deutsch. Arch. f. Klin. Med.*, Vol. 41, p. 323, 1887), considered that there was a rough correspondence between the temperature and leucocyte curves and that the leucocytosis was greatest at the severest stage of the disease. He also noticed that the leucocyte curve did not reach normal at the same time as the temperature, but took two or three days longer.

Boekman (*Arch. f. Klin. Med.*, Vol. 29, p. 491), asserted that the leucocytes and temperature bore a relation to one another.

Kikodse (*Path. Anat. d. Blut. b. Croup. Lung*, Inaug. Diss., Petersburg, 1890), thought that leucocytosis is absent only in fatal cases, that leucocytosis commenced before the lung is attacked and falls to normal with the crisis.

Von Jaksch (*Cb. f. Klin. Med.*, Nov. 5, 1892) attempted by giving leucocyte-increasing drugs to combat the fatality of those cases in which leucocytosis was absent, but he was disappointed in his results.

Rieder (*Beit. z. Kennt. d. Leucocytos*, Leipsic, 1892), found that there is no correspondence between the temperature and leucocyte curves, that the fall of leucocytes generally precedes the fall of temperature but that the temperature reaches normal several days before the leucocytes; that in cases of delayed resolution the number of the leucocytes remained elevated, and in cases ending by lysis fell to normal more slowly than in those cases ending by crisis. In one case he found leucocytosis six hours after the chill. In the majority of cases the polynuclear elements were markedly increased, but he did not think that there was any relation between the amount of leucocytosis and the extent of lung affected, it depending rather on the intensity

of the infection, and the degree of resistance of the subject. This latter observation was shortly afterwards supported by Ewing.

V. Limbeck (*Grundriss z. Klin. Path. d. Blutes.*, Jena., 1892), said that leucocytosis disappears with the fever; also that a steady rise in the leucocytes foretells a fatal ending. He also found leucocytosis on the first day of the disease.

Laehr (*Berl. Klin. Woch.*, No. 36 and 37, 1893), noticed that the temperature reached normal before the leucocytes. He found no correspondence between the leucocytosis, temperature, and amount of lung affected, but thinks that continued leucocytosis after the temperature has dropped to normal signifies delayed resolution. In one of his sixteen cases he found leucocytosis eight hours after the chill.

Rovighi (*Arch. Ital. d. Clin. Med.*, No. 3, 1893), observed that the leucocytosis is highest during the period of fall of temperature.

Cabot (*Bost. Med. and Surg. Journ.*, Vol. 130, No. 12), states that of seven cases which showed no leucocytosis six ended fatally. He also records that in the Massachusetts General Hospital of thirty-two cases of absent leucocytosis thirty died (*Clin. Exam. of Blood*, 1900). In one of the seven cases which ended in recovery there was a rise of leucocytes towards the end of the disease. He also states that there is no relation between the leucocytosis, the degree of severity of the case and the amount of lung affected.

Ewing (*N. Y. Med. Journ.*, Dec. 16, 1893), concludes that the greater the amount of lung involved the greater the leucocytosis; that in fatal cases there is no leucocytosis, and that a well-marked leucocytosis indicates a severe infection, absence of leucocytosis a grave prognosis.

Tchistovitch (*Arch. d. Sc. Biol. Imp. Inst.*, Petersburg, vol. 2, No. 5, 1893), in one of four fatal cases saw no leucocytosis.

Bieganski (*Deutsch. Arch. f. Klin. Med.*, vol. 53, 1894), studied the relative proportions of the various forms of leucocytes; 80 to 90 per cent. were polynuclears, eosinophiles being practically absent. Just after the crisis, the polynuclears fall below 60 per cent., and eosinophiles begin to appear. In fatal

cases, the polynuclears are reduced to 50 per cent., or below. The mononuclear elements are unaffected.

Billings (Johns Hopk. Hosp., Bulltn., Nov., 1894), examined twenty-two cases, ten of which were fatal. Only one of these showed complete absence of leucocytosis during the entire course of the disease. In six cases there was absence of leucocytosis at some period of the disease; but he found that the continuous absence was the exception, not the rule. The conclusions arrived at by Billings from the examination of his twenty-two cases were as follows:—(1). There is no correspondence between daily temperature and leucocyte curves during the febrile period. (2). That in cases where temperature falls by crisis, the leucocyte curve begins to fall within a few hours of the same time, but takes about forty-eight hours longer to reach normal. That in cases ending by lysis the two curves fall together, the temperature always reaching normal first. In cases of delayed resolution the leucocytes may remain elevated for days. (3). In the majority of cases the leucocyte curve rises during the period of fall of temperature; this is only transient, and is soon followed by a fresh fall. (4). That the correspondence of lung invasion and amount of leucocytosis is a very rough one. (5). The fatal cases may show a presence or absence of leucocytosis. (6). In severe cases, the continuous absence of leucocytosis is unfavourable to the prognosis. (7). The leucocytosis of pneumonia is a pure leucocytosis; *i.e.*, solely an increase of the polynuclear elements. In cases showing no leucocytosis the blood condition is normal. (8). The presence or absence of leucocytosis only shows the virulence of the bacterial poison.

Billings devoted most of his attention to the total leucocytosis in a large number of his cases, performing two blood-counts per diem. He draws his conclusions as to the variations of the different types of leucocytes from twenty counts made in different cases. In three counts made in cases where no leucocytosis existed, he found the blood normal.

Ehrlich and Lazarus (Die Anæmie). The increase of leucocytes usually continues up to the crisis, and this gives place to a diminution of the leucocytes until a subnormal number is

reached. They also agree that the absence of leucocytosis influences the prognosis unfavourably. Talking of post-febrile eosinophilia, Myers (Eng. Trans. of Ehrlk. and Laz., p. 152), says:—"At the height of most acute infectious diseases, with the single exception of scarlet fever, the eosinophiles undergo a relative decrease, and may even disappear entirely. In the post-febrile period, however, abnormally high values for the eosinophiles are often found, or even a well marked eosinophile leucocytosis, which generally attains but a moderate degree."

Ewing (Clin. Path. of the Blood, 1901) agrees with Rieder that the leucocytosis of pneumonia is not determined by the height of the fever, or the extent of the exudate, but depends on the intensity of the infection and the degree of resistance of the subject. He says that leucocytosis appears very early in the course of the disease. In one of his cases he found 25,000 cells within four hours of the chill. He does not think that extension of the disease to other lobes affects the leucocytosis at all considerably. In a case in which bronchial breathing passed successively up one side of the chest and down the other he found a rather uniform and high leucocytosis. He is of opinion that a few hours before or after the crisis the leucocytes begin to diminish rapidly, sometimes falling from a high to a normal figure within twenty-four hours, after which there are commonly some slight oscillations. During lysis the reduction of leucocytosis usually keeps pace with the temperature. The amount of the leucocytosis he considers was influenced by the extent of the exudate. The records of his cases certainly tend to show this; thus, in sixty-three cases with one lobe affected average leucocytosis 20,000, in twenty-four cases with two lobes affected average 22,700, in twelve cases with three lobes affected 25,000, in one with four lobes affected 27,000, and in one case in which there was bronchial breathing over the entire back of chest 32,000.

*Types of leucocytes in pneumonia.*—The chief work on this subject has been done by Türk (Klin. Unters. d. Ver. d. Blut. Infect. acut. Leipsic, 1898) and by Stienon (Recherches. sur la leucocytose dans la Pneumonie aigue. Brux., 1895). Other writers have contributed towards it but none so completely as

Türk and Stienon. Bieganski's work has already been referred to. Türk found that in the leucocytosis of pneumonia there is an increase of the polynuclear cells. In a large number of cases he found that they rose above 90 per cent., and in one case he counted as high as 96·5 per cent. He states that this proportion may be seen even when the leucocytosis is slight, but is more marked with a high leucocytosis. The lymphocytes are at the same time diminished in proportion, but usually the large mononuclear cells are present in considerable numbers. The eosinophiles are much diminished in number, and sometimes disappear altogether. After the crisis the polynuclear cells diminish rapidly to a high normal proportion, at which they may remain for several days. As the polynuclears diminish towards their normal, so the lymphocytes increase in proportion also after a few days. At this period the large mononuclear cells are usually over-abundant, being 16 per cent. in one of Türk's cases. Eosinophiles reappear in scanty numbers on the day before the crisis or occasionally a day earlier. After the crisis eosinophilia distinctly appears in a large proportion of cases, but not invariably. Myelocytes were found by Türk in nearly all cases, being most abundant at the time of defervescence, and in one case reached a proportion of 11·9 per cent. in 8,000 cells.

Stienon comes to the conclusion that in most cases the number of leucocytes varies considerably, the increase as a rule disappearing with the increase of fever. He considers that in the febrile stage the polynuclear cells predominate, the eosinophiles being very scarce. After the crisis the lymphocytes increase greatly. In cases of delayed resolution the polynuclears remain increased. As soon as the polynuclears begin to diminish the eosinophiles increase, this increase occurring early in mild cases and being slight or wanting in cases of suppuration. He also describes a case in which the young forms of cells predominated. This observation was confirmed by Cabot, who described a case occurring in the Massachusetts General Hospital, where the small lymphocytes made up 66 per cent. in a leucocytosis of 94,600.

Referring to the large mononuclear cells in pneumonia, Klein says they may sometimes be distinctly increased, whereas Jez makes the remark that they are never entirely absent. Klein also remarks that during the prolonged lysis in pneumonia, a relative high proportion or increased number of lymphocytes is usually observed. Ehrlich states that in the initial or persistent hypoleucocytosis observed in infectious diseases (pneumonia, diphtheria) the remaining cells are largely lymphocytes.

Pick (*Arch. f. Dermat. und Syph.*, Vol. 25, p. 63), noted that pneumonia complicating typhoid fever caused no leucocytosis.

*Prognosis in cases of absent leucocytosis.*—Although generally considered fatal, this does not seem to be invariably the case. Out of fifty-seven severe cases without leucocytosis collected by Ewing and reported by Halla, Hayem, Laehr, Ewing, Sadler, Bieganski, Zappert, Türk, Billings, Cabot, only forty-four were fatal, although all of them were unusually severe cases.

#### CLASSIFICATION AND NORMAL NUMBERS OF LEUCOCYTES, ETC.

*Classification.*—That of Ehrlich is the classification most universally adopted, and since Ehrlich's triacid stain was used in preparing all the films here recorded, and especially as the classification depends on the staining reactions of the cells, I have deemed it advisable to follow his mode of distinguishing the different types of white corpuscles.

(NOTE.—The figures attached to the individual varieties represent the percentages to be found in normal blood.)

Polynuclear leucocytes	...	...	70 per cent. to 72 per cent.
Lymphocytes	...	...	22 per cent. to 25 per cent.
Large mononuclear leucocytes	..		1 per cent. approximately.
Eosinophiles	...	...	2 per cent. to 4 per cent.
Transitional	...	...	2 per cent. to 3 per cent.
Mast. cells	...	...	0.5 per cent. (maximum).
Myelocytes	...	..	do not occur in normal blood.
Nucleated red cells	...	...	rarely occur in normal blood.

Normal number of red cells per cubic millimetre :—

Males	...	...	5,000,000 to 6,000,000.
Females	...	...	4,500,000.

Normal number of leucocytes per cubic millimetre :—

Limbeck	... 8,000 to 9,000.
Rieder ...	... average, adults 7,680 (9,600–4,800). children 9,960 (12,400–7,200).
Reinert ...	... Time, 6 a.m., 5,125. 4 p.m., 8,262.

*Methods.*—To obtain the blood the lobe of the ear was pricked with a fine lancet-pointed needle after thoroughly cleansing the skin with absolute alcohol. All blood required was taken from the ear within a few minutes, that is to say, the blood taken for the films was almost the same drop as that taken for the red and white cell estimation and the hæmoglobin. As far as possible the blood in each case was procured at the same hour each day, but this on some occasions was impracticable. The times of taking blood are given in each case in the tables below the charts. The apparatus used for estimating the numbers of the two kinds of corpuscles was the Thoms-Zeiss apparatus with Hayem's fluid for the red corpuscles and a 0·5 per cent. solution of glacial acetic acid coloured with a little gentian violet for the white corpuscles. It is unnecessary for me to detail the manner in which this instrument is used, as it is so well known, but it would not be superfluous to mention the special precautions which were taken to ensure accuracy in the examinations which were undertaken.

The blood having been secured in the pipette was well shaken for the space of one minute with the diluting fluid, after which the undiluted blood in the capillary tube of the pipette was blown out. The ends of the tube were closed with a thick india-rubber band and placed aside for further examination, when before each drop of blood was put on the counting chamber, a further vigorous shaking was given for the same length of time. The blood was always examined on the same day as it was procured. For the sake of convenience three counting chambers were used, as time was thereby saved, a consideration of no mean importance when five or six blood-counts of both red and white corpuscles have to be finished in one day.

No drops were accepted on the counting chamber unless Newton's rings were visible, and a damp atmosphere seemed to favour their production. Never less than five separate drops were counted for estimation of the leucocytes, and the average of these was taken as the result. Two or three drops were taken of the diluted red corpuscle fluid, and 1,000 to 1,200 cells were counted in every case, the average being taken of the number of cells in each sixteen small squares of the counting chamber. A Leitz one-sixth with objective was used for the examination, and a mechanical stage for moving the counting chamber across the field of the microscope.

*Estimation of Hæmoglobin.*—For this purpose, a small and convenient instrument, known as Reichert's hæmoglobinometer, was used. In principle, it is a modification of Gower's instrument, with the advantage of a glass standard of comparison instead of the jelly of Gower's instrument. It is said not to fade as readily as the jelly standard of Gower's. It is a small and very portable instrument, and in its results compares favourably with Haldane's apparatus (carb-oxy-hæmoglobin principle), which, I believe, is recognised as the most efficient of modern hæmoglobinometers.

As far as possible, the same artificial light was used in order to obtain results as uniform as possible.

*Preparation of films.*—Films were made on coverslips, which had been previously cleaned by boiling for some minutes in nitric acid (chromic acid was found to leave a yellow stain), and keeping ready for use in absolute alcohol. When a film was to be prepared, the coverslip was dried and polished with a silk handkerchief. A pure rice, rather stiff, cigarette paper was used to catch the exuded drop of blood, and this being applied to the coverslip, and allowed to spread in an even film over it, was then steadily drawn over the surface of the coverslip, which was held in a very simple and inexpensive form of clip. This latter, made for me by C. Baker, optician, High Holborn, from whom they may be obtained, consisted of a piece of sheet rubber about one-eighth of an inch thick, cut half way through its thickness, and then bent over in the form of a hinge. One side of the hinge,



which was made broader than the other, was mounted on a piece of wood, also about one-eighth of an inch thick, and an ordinary paper clip was used to approximate the two surfaces of rubber joined at the hinge. The coverslip is held between the two surfaces of rubber, and cannot move, as the cigarette paper is drawn over its surface. The clip I used would hold six coverslips, of varying thicknesses, and all would be held without fear of slipping.

The films so made were placed in small envelopes, and put aside until it was convenient to fix and stain them.

*Fixing the films.*—For this purpose dry heat was found to give the best results, a small copper oven with a thermometer to register the temperature and some means of limiting the supply of gas to the Bunsen flame was found to be most convenient. When the films were to be fixed, the oven was heated up to a temperature of  $140^{\circ}\text{C}$ . The films were placed on pieces of filter paper and then deposited in the oven. (NOTE.—It is best to heat the oven a little above the temperature required, as when the door is opened to put the films inside, the temperature invariably drops two or three degrees.) An average film was left in the oven for the space of two minutes exactly. If the film was spread rather thinner than the average, one minute and a half instead of two minutes was used, but in no case except when the leucocytosis was exceptionally great was this time diminished. When performing experiments in order to find the length of time required for fixing films which were to be stained with the triacid stain, I had occasion to stain some films from a case of leucæmia in which the leucocytosis was 200,000 per cubic millimetre, and could only get them to stain correctly by using the very smallest amount of heat, namely, by passing them once or twice rapidly through a flame, but for ordinary films with a leucocytosis up to 50,000 it was found that from one and a half to two and a half minutes at  $140^{\circ}\text{C}$  was a safe time and temperature for fixing them. Cabot, using Ehrlich's triacid stain, gives a much higher temperature and gradually raises the temperature of the films to the required point. This method gives inferior results, the staining of the nuclei being very poor, but

possibly with the brand of stain that Cabot used, he did not find this so, as undoubtedly the length of time required for fixing varies with the brand of stain in use. The time and temperature above stated coincides more with that suggested by Ehrlich himself.

*Staining the films.*—As stated before, the stain used throughout for all those films, the results of which are recorded in the tables below the charts, was Ehrlich's triacid mixture, and the most important point is the method of its preparation, for, unless made up correctly the time of fixing must be much prolonged. Various ready mixed brands of stain were obtained from several dealers in London, and with all of them, including the mixture prepared by Grubler, I was unable to obtain good results, except by very prolonged fixing, and even then the results were poor, the nuclei staining feebly. In making the stain the following points were observed :—

(1) Grubler's dry stains were used, and saturated solutions of these were prepared some days before the triacid mixture was made up. Distilled water was used.

(2) The formula used was that recommended by Cabot and the method of preparing also that advised by him with the following additions :—

(a) When the orange G. and acid fuchsin had been mixed, the mixture was exposed to the air for about half an hour.

(b) The saturated solution of methyl green was filtered and then added drop by drop to the mixture of orange G. and acid fuchsin.

(c) The glycerine was added drop by drop after the absolute alcohol and water had been added.

Any departure from the above method, even though the right proportions of the dry stains were used, did not give a successful triacid stain.

Many people have given up the triacid stain in despair as they were unable to obtain good results with it. The whole secret of successful results appears to depend on its method of manufacture. Therefore, it is important to follow out carefully

all details necessary for its preparation, and when once a good stain has been made up it is exceedingly simple to use.

The stain was poured on to the films as soon as they were taken out of the oven (while still hot) and was left in contact with them for about ten minutes. If a less time than ten minutes was given the nuclei were not fully brought out. It appeared to be impossible to overstain. The films were then washed in water, dried between filter papers, and mounted in the usual way in Canada balsam.

In counting the films, a Leitz one-tenth dry objective was used, but if any cells were seen of doubtful variety the one-twelfth oil-immersion was substituted and showed up the details a trifle better than the tenth, but was not so convenient to use. As will be seen by the tables, in most instances 500 cells were counted, a less number only being enumerated when the films were inferior. In some cases 1,000 cells were counted, but I have convinced myself that 500 is quite sufficient for all practical purposes.

#### EXPLANATION OF CHARTS AND TABLES.

The charts although looking somewhat complex at first sight on account of the various curves being superimposed, as the eye becomes accustomed to the distinguishing lines this difficulty is soon overcome. It will be seen that the temperature curve is drawn up in the usual manner on a four-hours chart. The leucocyte curve is drawn up over this and the height to which the leucocytes rise each day can be read off by following the straight lines of the chart to the figures under the words "LEUC." In the same manner the curves of the polynuclear cells, lymphocytes, and eosinophiles show their respective percentages each day by following the lines of the chart to the figures under the words "POLYNUC," "LYMPH," "EOSIN," and in those charts where myelocytes are represented, under the word "MYELOCYT." It has not been thought necessary to draw curves for the large mononuclear and transitional cells, as in cases where these are much increased in numbers, the symmetry of the figure which the polynuclear and lymphocyte curves together make across the chart is disturbed. Of course in cases

where the eosinophiles are much increased, the symmetry is also disturbed. The absolute numbers per cubic millimetre of each individual type of cell is given in the tables, in brackets, at the side of the percentage numbers. In the lower part of the tables, under the heading "OTHER CELLS, NOTES, ETC.," is given an account of those cells which I have been unable to classify, also the chief physical signs which existed on the day of taking blood, and other conditions which were likely to affect the blood. All blood was taken at periods when digestion-leucocytosis could be avoided. The numbers of the red cells are a guide as to whether the blood is concentrated or otherwise.

CASE 1. *Pneumonia, right base*.—E. H., æt. 16, admitted under the care of Dr. Pitt, October 24th, for dyspnoea and pain in the right side. History of case.—On October 18th, patient suffered from a cold in the head. On October 20th, she felt very much worse, began to cough, and complain of pain in the right side. She felt very chilly, and said that it hurt her right side when she swallowed. On October 21st, she was no better, and consequently kept her bed. The cough and pain in the side had increased in severity, and in addition, she was very short of breath. She had not vomited, and her bowels had been regular, but only with the aid of liquorice powder. *Condition on admission*.—Pulse 100, temperature 102·8°, respiration 34. Patient is a well-nourished girl, but very pale. She lies on her right side, and complains of pain under the right breast, which is worse on deep inspiration and sudden movement. Respiratory system.—Right side of chest does not move so well as left during respiration. There is no absolute dulness, but impaired resonance over the lower five spaces behind, on the right side, and over this area bronchial breathing, crepitating râles, and bronchophony can be heard. Left side of chest normal. She is coughing up a characteristic rusty sputum. Urine 1024; trace of albumen. Progress.—October 24th. Dulness more absolute, and signs occupy a larger area behind. She is also in much more pain. October 26th. The area of dulness behind has diminished. Patient has been put on salicylate of soda. October 27th. Crisis occurred to-day. During the crisis, she sweated a considerable amount. Later in the day she began to feel hungry. October 28th. Râles have disappeared, also the rusty sputum has ceased. October 31st. There is still deficient note over the right base behind, together with slight bronchophony in right axilla. The cough has ceased.

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CASE 2. *Pneumonia, right base*.—T. C., æt. 22, admitted under the care of Dr. Washbourn, November 19th, for feverishness. History of case.—On day before admission patient was seized suddenly, while on duty as a night policeman, with a definite shivering attack. Soon after he commenced to vomit. He went off duty and saw the divisional surgeon who sent him up to the hospital. He was thought to be suffering from influenza and was told to come up next day if no better. Vomiting continued throughout that day and on the morning of the day of admission. A cough and pain in his

right side behind with a very severe headache developed on the day he was admitted to the hospital. No diarrhoea. No soreness of throat. *Condition on admission.*—Pulse 116, temperature 104°, respiration 32. Patient was a muscular young man. He stated that he felt very ill and that he had a very bad headache. He was also inclined to wander in his speech. No rash seen on body. He had some cough but brought up no sputum. Respiratory system.—Only physical signs were a doubtfully impaired note at right base. Urine 1028; trace of albumen; chlorides, only a faint trace. Other systems normal, but tongue was slightly furred. Progress.—November 20th. Patient was so restless during the night that morphia had to be administered. No physical signs in the lungs. November 21st. There was a constant rhonchus at base of right lung in front. The abdomen was more tumid. Patient had a very restless night. Spleen could not be felt. As an examination of the blood showed a considerable leucocytosis (see tables) a diagnosis of pneumonia was made although there were no physical signs in the lungs. It had been doubtful whether it might not be a case of typhoid fever. November 22nd. Entry of air at base of right lung from angle of scapula downwards was not so good as on the left side. A very few fine rustling râles were heard at angle of right scapula. November 23rd. Marked physical signs heard at angle of right scapula, namely, impaired resonance, deficient entry of air, consonating râles, bronchophony, and bronchial breathing. Some rusty sputum was coughed up. November 24th. Bronchial breathing marked from middle of angle of right scapula down to base of right lung. In front there was well marked distant bronchial breathing all over right side with numerous small consonating râles. Sputum had now become prune-juice in colour. November 25th. Crisis took place. Bowels were open four times and he sweated so much that his shirt had to be changed three times. November 26th. Pain in right side reappeared and he could not sleep. November 27th. Reduc crepitations over whole of right lung behind. Bronchial breathing absent. Dulness had given way to an impaired note. Pain had ceased. Sputum scanty and yellowish in colour. November 28th. Patient was feeling quite well in himself. Still a few consonating râles at right base and over same area there was deficient entry of air. Still marked diminution of chlorides in urine. November 30th. All physical signs had cleared up and patient was convalescent.

CASE 3. *Pneumonia, left base.*—B. W., æt. 17, admitted under the care of Dr. Washbourn, December 13th, for pain in left side and shivering. History of case.—Patient was out late on the night of December 8th, when he got wet through and thought that he caught a chill. He has been in bed since and had several shivering fits. The only food he has taken since that time has been a little milk. On the day of admission he felt a sharp pain in the left side. *Condition on admission.*—Pulse 96, temperature 102.6°, respiration 28. Patient was a healthy young man; his cheeks were very flushed and pupils dilated; bowels had only been open of late with the aid of liquorice powder. Respiratory system.—There were signs of consolidation at the left base, namely:—Increased tactile vocal fremitus, impaired note, bronchial breathing and a few consonating râles. Large area of very marked pectoriloquy limited to left base, whole of lower lobe. He was expectorating typically rusty sputum. Urine sp. gr. 1020, no albumen, diminished chlorides. Other systems normal. December 14th. Very delirious during the night, but pain was better. Physical signs not so

marked. Pectoriloquy very much less, but there were many more râles. Bronchophony not so well heard. December 15th. Still very delirious during the night. Dulness not nearly so marked. Area over which signs of consolidation had existed diminished. Only slight amount of rusty sputum still present. December 16th. Physical signs had all disappeared except for a few redux crepitations. No rusty sputum. December 17th. Patient convalescent and was allowed up.

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CASE 4. *Pneumonia, right apex.*—F. C. H., æt. 10, admitted under the care of Dr. Pitt, October 18th, for fever, pain in the side and cough. Previous illness.—Pleurisy two years ago. History of case.—On the morning of October 16th, while at school, he complained of headache and pain in the stomach and right side. On October 17th he felt tired and heavy; he had also vomited after some milk. On the morning of October 18th he was hot and feverish, he also coughed a good deal, so he was brought up to the hospital. *Condition on admission.*—Pulse 100, temperature 103.4°, respiration 30. Patient has a typical pneumonic appearance, bright eyes and flushed cheeks, etc. He has a short cough and brings up some slightly yellow sputum. The only physical signs to be heard in the chest are numerous râles and rhonchi in front and behind. Urine sp. gr. 1028. Trace of albumen, chlorides not diminished. Other systems normal. Progress.—The temperature was thought to be too high for a case of bronchitis only, and pneumonia was consequently diagnosed. October 20th. Vesicular murmur over the right chest not so audible as on the opposite side. October 21st. Over the right apex behind and in the right axilla there is an impaired note, fine crepitations, faint bronchial breathing and pectoriloquy. L'nemcocci were found in some of the sputum obtained yesterday. Apical pneumonia was diagnosed. October 22nd. Patient's temperature fell by crisis this morning. There is an impaired note at the right apex and in the right axilla. Bronchophony and pectoriloquy have disappeared, but there are still a few fine râles. October 24th. Diminished vesicular murmur over right lung is the only abnormal physical sign to-day. October 28th. Patient is convalescent. Lungs have cleared up.

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CASE 5. *Influenzal pneumonia.*—H. S., æt. 15½, admitted under the care of Dr. Pitt, November 5th, for cough and fever. History of case.—Patient returned from her work on November 2nd, apparently in perfect health. On the following day, November 3rd, she was noticed to have a troublesome cough. After dinner she started vomiting and continued to do so many times. She expectorated a great deal and had a bad frontal headache and pains between the shoulders. Her cough kept her awake at night and she was unable to retain any food. The following day, November 4th, she still vomited and the other symptoms were exaggerated. She was brought up to the hospital, and a rub was heard at the left base. On November 5th, she was again seen and admitted. *Condition on admission.*—Respiratory system.—Front of chest, rhonchi could be felt and the tactile vocal fremitus was much less marked on the left side than on the right. Resonance unaltered. Sticky râles could be heard all over the chest on both sides but chiefly on the left and more numerous at the base. Rhonchi of all varieties could be heard on the left side, but mostly sibilant on the right. Fine

crepitations could be heard in the left axilla and a doubtful rub at the left base. Voice-sounds unaltered. Back of chest, tactile vocal fremitus less marked on the left side. Right side seemed a little less resonant and the vesicular murmur was poor at the left base. Otherwise signs same as in front. Sputum, thick greenish yellow. Cough was very troublesome. Other systems normal but tongue was furred, and breath rather foul. November 6th. Physical signs remain the same. November 7th. Râles and rhonchi less numerous on both sides behind and in front. Patient seems very well in herself. Crisis took place during latter part of yesterday and to-day. November 10th. Vesicular murmur is not heard so well at base as elsewhere, also at right apex the vesicular murmur is not so good behind, but no râles could be heard. Patient is quite convalescent. On November 11th, a few râles and rhonchi were still heard.

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CASE 6. *Pneumonia (right lung). Absent leucocytosis. Death.*—R.M., æt. 24, admitted under the care of Dr. Pitt, October 22nd, for fever and pain in right side. History of case.—Patient did not feel well on October 19th. On October 20th, she saw a doctor who diagnosed pleurisy and pneumonia. She was sent to bed, but as she did not improve and the pain in the side got worse she came up to the hospital and was admitted. *Condition on admission.*—Pulse 120, temperature 104·6°, respiration 36. Patient is a healthy young female. She has a short frequent cough which intensifies the pain in the side. She is bringing up a viscid sputum, rusty in colour. Urine 1020; trace of albumen. Marked diminution of chlorides. Respiratory system.—The whole of the right lung behind is dull, with increased tactile vocal fremitus, bronchial breathing, bronchophony, pectoriloquy and fine râles; especially at the extreme base. There is a distinct rub in the right axilla and an impaired note up to the fifth rib, with some fine râles. Pulse soft and rapid. Other systems normal. Progress.—October 23rd. Patient passed a fair night. Physical signs unaltered. October 24th. Patient feels a little better. Respirations are still 40 and she is somewhat cyanosed. Practically the whole of the right lung is involved. There is bronchial breathing in front on the right side as far as the second rib, with impaired note. Rub in axilla still audible. Taking the age and constitution of the patient and the fact that the consolidation was limited to one side a favourable prognosis was given, but the result of the blood examination (see tables) reversed the opinion. October 25th. Cyanosis less. Patient passed a restless night, being in and out of bed several times. This morning she wanders and complains that she cannot see people. Physical signs remain the same. October 26th. Patient is much worse, is very restless and had practically had no sleep during the night. She talks and mutters incoherently to-day. Cyanosis more intense. Physical signs unaltered. Pulse is very weak and rapid. October 27th. At 4.30 a.m., she became very much worse and intensely cyanosed. Pulse rapid and intermittent. Oxygen gas, and strychnine hypodermically. She rallied for a time but pulse again became very intermittent and she suddenly stopped breathing at 9.30 a.m. Post-mortem examination showed that the whole of the right lung was consolidated except for a small portion at the apex, which was airless.

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CASE 7.—*Double pneumonia, both lower lobes; delayed resolution, otorrhœa.* A. H. S., æt. 6, admitted under the care of Dr. Pitt, November 8th, for

cough and pain in side. Previous illness.—Has had a discharge from his right ear two years ago, also had measles last year. History of case.—Patient was quite well until November 4th, when he complained of a headache. On November 5th he was hot and feverish, also coughed a good deal. He was seen by a doctor, who said he was suffering from bronchitis. The doctor made a further diagnosis of pleurisy on November 7th, and discovered that pneumonia was present as well on November 8th. Accordingly he was sent to the hospital on that day. He has not been sick and the bowels have not been open since November 6th. He has also complained of pain in both sides of chest since the morning of November 7th. *Condition on admission.*—Pulse 134, temperature 100·8°, respiration 48. Patient is a well-nourished boy. He lies on his left side and when disturbed has a short and frequent cough. There are four vaccination pustules on his left arm (about ten days old). Respiratory system.—Patient is not expectorating any sputum. Both sides of chest move equally. Normal resonance at apices in front. Impaired notes in both axillæ. Dulness at both bases behind from seventh space. A to-and-fro rub can be heard in right axilla and at both bases. There is also deficient vesicular murmur, bronchial breathing, bronchophony, pectoriloquy and some crepitations at times over the same area, also a rub. The vesicular murmur at apices behind not very good, and some moist sounds are heard. Urine, sp. gr. 1032; trace of albumen; some diminution of chlorides. Other systems normal, but tongue is furred and some sordes on the lips, November 10th. Physical signs remain the same, namely, those of consolidation at both bases. November 12th. Crisis took place during yesterday and to-day. The dulness at both bases, with bronchophony, etc., still exist. November 13th. Otorrhœa appears in the shape of a slight muco-purulent greenish discharge from both ears (for effect on leucocytosis see tables). Physical signs of consolidation unaltered. Patient feels quite well in himself. November 14th. Physical signs are not clearing up. Same physical signs exist. Ears examined showed small perforation in drum of left ear. In the right ear there is a very large perforation, only the upper portion of the mucous membrane being left, and looking as if the perforation was an old one with some more recent trouble. No pneumococci found in the pus of either ear. November 18th. Physical signs much the same as above. Dulness at both bases, bronchial breathing, râles, bronchophony, pectoriloquy, etc. No rub heard. November 21st. Dulness at both bases less marked. Still some bronchial breathing, and pectoriloquy at angle of left scapula, also consonating râles, especially after coughing. November 24th. Dulness not so marked, only an impaired note at both bases. Bronchial breathing has disappeared but there are still consonating râles after coughing. November 29th. The right base has completely cleared up and there is only an impaired note at left base. Discharge from right ear has ceased. The left ear still discharges freely. December 3rd. Patient got up to-day for the first time. December 4th. Lungs have quite cleared up now. December 6th. Patient discharged to-day. Lungs have quite cleared up, but left ear is still discharging freely.

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CASE 8. *Pneumonia (left, lower and middle lobes). Delayed resolution. Upper right lobe (slight)*—B. G., æt. 8, admitted under the care of Dr. Pitt, October 28th, for feverishness. History of case.—Fourteen days before admission patient had a bad cough and cold. On the night of October



27th, she became suddenly ill, was sick twice and could not get her breath; she also appeared to be very feverish. She was brought up to the hospital on the afternoon of October 28th. During October 27th, her cough was much worse but she did not bring up any phlegm. *Condition on admission*.—Pulse 160, temperature 103°, respiration 56. Patient is a pale and nervous child. She has a dry irritating cough, but brings up no sputum. Respiratory system.—Both sides move equally; no dulness; puerile breathing heard all over the chest; harsh and loud bronchial breathing over upper three spaces in front of right chest. No cyanosis. Urine, sp. gr. 1034; trace of albumen. Diminution of chlorides doubtful. Other systems normal. Progress.—October 29th, bronchial breathing over right upper lobe. Pulse 156. October 31st. Patient is very drowsy. Bronchial breathing and impaired note is now present at back of upper lobe (right). Fine crackling râles over left base behind. November 1st. There is dulness over the whole of left lower lobe behind and also an impaired note in axilla. Bronchial breathing, ægophony and crackling râles over the same area. The whole of left lower lobe appears to be solid. A rub is heard in axilla. November 2nd. Pain in left side on coughing. Respirations 56, and typically pneumonic in character. There is now impaired note, râles and bronchial breathing over the middle lobe. November 6th. Temperature dropped to 98° but breathing is still rapid and she seems generally in a worse condition. Physical signs over left chest unaltered. The bronchial breathing in front of right apex cannot be heard now, but the percussion note is still impaired. November 8th. Patient feels much better and is playing with her toys. November 10th. Physical signs over left chest are not clearing up. November 12th. Left base more resonant, râles have nearly all disappeared, but bronchial breathing and dulness still exist over middle lobe. November 15th. Physical signs are clearing up quickly. November 19th. The note obtained over the back of left chest is only very slightly impaired now. There is no bronchial breathing present and only a few râles. November 21st. Physical signs have all cleared up except for a few fine mucous râles over left middle lobe behind. November 25th. Discharged to convalescent home.

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CASE 9.—*Pneumonia, left base. Typhoid*.—T. G., æt. 9, admitted under the care of Dr. Washbourn, November 21st, for fever, cough and diarrhoea. Family history.—On the day of admission a sister of the patient was convalescing from typhoid fever. She had been admitted on October 29th with signs of pneumonia. History of case.—On November 17th patient complained of headache and pain in the stomach, also that his legs ached. He was put to bed and seen by a doctor, who said he was suffering from pleurisy and bronchitis. He was restless and feverish, and on November 20th was sick. Bowels had been open daily, and on the day before admission they were open six times. *Condition on admission*.—Pulse 104, temperature 103·8°, respiration 48. Patient looked physically a strong lad, but was rather small for his age. His skin was hot and dry. No herpes. He was inclined to wander in his sleep and was very restless. Respiratory system.—Cough occasional and short. Both sides of chest moved equally. Dulness at left base from spine of scapula downwards. Impaired note in left axilla. Mucous râles and rhonchi heard over both lungs. At left base there was bronchial breathing, bronchophony, pectoriloquy and fine crepitations.

Alimentary system.—Lips, some sordes. Tongue furred, brown and dry. Abdomen.—Some tenderness. Spleen not felt. No spots. The motions were fluid, light yellow in colour, foul-smelling, and contained some undigested curds. Urine.—Sp. gr. 1018; trace of albumen; no diminution of chlorides. Pneumonia at left base diagnosed. Progress.—On November 23rd abdomen distended and tender on pressure. A typical rose spot seen on chest. Blood examined for Widal's reaction gave a positive result. Pneumonia together with typhoid fever diagnosed. November 25th. Physical signs at left base unaltered, except that no bronchial breathing was to be heard. November 27th. Patient was sleeping better and did not wander in his speech at night. Numerous râles and rhonchi all over front of chest. November 30th. Temperature fell to subnormal and patient was much brighter. Tongue was clean and moist. Still numerous râles and rhonchi all over chest and in axilla, and at left base the râles were of a very consonating character. December 3rd. Râles and rhonchi in chest very much less, in fact, all physical signs were rapidly clearing up. December 4th. All physical signs had disappeared, except for a little impaired resonance at left base and a few râles and rhonchi on coughing. December 5th. A few small boils appeared on sacrum. Fomentations were applied. December 9th. Patient convalescent.

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CASE 10. *Pneumonia complicating Henoch's purpura. Empyema. Death.*  
 —I. M. D., æt. 6, admitted under the care of Dr. Washbourn, November 15th, for hæmorrhage from the bowel and purpuric spots on the legs. History of case.—On November 11th, patient complained of pain in legs, thighs and abdomen. On November 13th, she passed a motion containing bright red blood. This continued until November 15th when her bowels were opened twenty-two times, the motions consisting chiefly of blood and clots. She was sent up to the hospital and admitted. *Condition on admission.*—Patient is a bright healthy-looking child. She does not look at all anæmic. There is a small cutaneous hæmorrhage on the right cheek, and over her buttocks, legs and thighs, chiefly on the flexor surfaces, is a purpuric eruption. Most of the spots are purple but some are of a bright red colour. Progress.—The diarrhoea and bloody stools continued severely until November 22nd, and on November 18th and 19th, she vomited persistently. There was no blood in the vomit. The purpuric spots which had nearly cleared up by November 21st, again appeared all over the feet, legs and buttocks with a few on the chest, but they were clearing up rapidly on November 30th, and patient was feeling very well. On December 1st she did not take her food well, but nothing was found to account for this. On the evening of December 2nd, her temperature rose to 102.4° and respirations to 48. She complained of pain in the right side of chest, but still nothing could be found to account for this. On December 3rd, distant bronchial breathing was detected at base of right lung behind, and on December 4th, there was definite bronchial breathing over lower six ribs behind on right side. There were also increased voice sounds heard all over lower lobe of right lung and over same area an impaired note existed on percussion. December 5th. Signs of consolidation appeared to be extending and involved middle lobe of right lung. The whole of lower lobe on left side gave impaired note on percussion and over it crackling râles and bronchial breathing were to be heard. Patient was slightly

cyanosed and had a good deal of pain. Her pulse was weak and rapid. December 6th. The note over lower and middle lobes on right side was still more impaired and over them harsh bronchial breathing and dry râles were to be heard. The signs on left side remained the same. Pulse was weak and she was a very bad colour. As the leucocytes had dropped to 2500 per cubic millimetre it was decided to give the patient nuclein in order to increase the number of leucocytes. December 9th. Impaired note on left side had extended upwards to level of fourth rib and note was impaired over the whole of right lung behind. December 12th. Patient much worse. Physical signs were not clearing up. Pulse very weak. December 15th. The note on the left side rather more resonant. No change in right side. December 17th. Patient seemed much better and temperature dropped to 99.4° but pulse was still very weak. Left chest much more resonant, but there were râles and bronchial breathing all over back of left chest. Behind the right chest the note was very impaired from fourth space downwards with bronchial breathing and râles over this area. December 19th. Left chest appeared to be clearing up but right chest remained as before. December 21st. Patient did not seem to be progressing. Note obtained over lower six spaces on right side quite dull. An aspirating needle inserted into this place did not show the presence of pus. December 22nd. Patient very much worse. Signs of meningitis appeared. Pulse became very weak and rapid, improving temporarily under stimulants until her death at 10.30 on December 23rd. Post-mortem examination.—Twelve ounces of pus found in the right chest. Whole of lower and middle lobes on right side were compressed, solid and airless. Upper lobe normal. Patches of broncho-pneumonia over lower lobe and lower part of upper lobe on left side. Upper part of upper lobe normal. Heart large. Recent vegetations on mitral valve. Small amount of clear fluid in pericardium. Brain.—Quantity of purulent fluid extending throughout meningeal cavity.

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Of these ten cases it will be observed that two were fatal and eight ended in recovery. Out of the eight cases which recovered, in six the temperature came down by crisis, namely, cases 1, 2, 3, 4, 5 and 7; one by lysis; and the other, a case complicated by typhoid fever, the temperature was of a typical typhoid character. Of the six cases in which the temperature came down by crisis, in case 5 the crisis was protracted and in cases 4 and 5 there was a post-critical rise of temperature. Of the other cases, one was a case in which there was no leucocytosis, and the other, which proved to be more of a broncho-pneumonia, complicating purpura, eventually ending in empyema. Both these latter died.

*The red corpuscles.*—All the cases, except case 2, showed a fall in the red corpuscles during the crisis, the average drop in numbers being 280,000 per cubic millimetre. After this temporary drop, it is noticed that the red corpuscles rose again during the

next few days in cases 1, 2, 3, 4 and 7; but in case 5 they fell a little further after the crisis and remained at that number. The average rise after the crisis in cases 1, 2, 3, 4 and 7 was 480,000 per cubic millimetre. In case 8, where the temperature fell irregularly, the corpuscles dropped during the fall of temperature, and remained down. In cases 9 and 10 the red corpuscles did not show much, except in case 10 the progressive diminution in numbers as the child got worse.

The chief value of a table showing the red corpuscles and the leucocytes at the same time, lies in the fact that the red cells by their numbers show whether the blood is concentrated or otherwise. One would rather expect to find an increase of red cells during the crisis on account of the sweating which most frequently takes place, but this does not appear to be so in the cases recorded above. Billings' charts, although excellent, would have been of greater value had he shown the numerical proportion of red corpuscles to leucocytes, as erroneous views may be taken of an increase of leucocytes if the blood is concentrated and its existence is not known.

Nucleated red cells were only seen in three out of the ten cases; namely, on one occasion each in cases 7 and 8, which were cases of delayed resolution, and almost every day in case 10, which eventually ended in empyema. The greatest number to which they reached in case 10 was 1·4 per cent.; and these were chiefly normoblasts. It will be remembered that Türk found chiefly normoblasts but occasionally megaloblasts in the cases examined by him.

*Hæmoglobin.*—The hæmoglobin figures in the charts are somewhat irregular. In cases 1, 2, 5 and 7 they were diminished during the crisis. In case 3 the hæmoglobin was increased during the crisis, but this figure was obtained by an independent observer, so that I am not absolutely certain of the accuracy of the hæmoglobin estimation in this case. In case 4 the hæmoglobin remained the same until after the post-critical rise of temperature, when it fell. In cases 2, 4 and 7 it rose again after this initial fall. Likewise in case 3, but in cases 1 and 5 it remained down. This initial fall during the crisis followed by a subsequent rise

was just what was found in the majority of cases to take place with the red corpuscles. So taking into account the fact that the estimation of hæmoglobin is only accurate to 5 per cent. with the present hæmoglobinometers which depend so much on individual acuteness of colour vision, I think it is safe to say that the hæmoglobin and red corpuscles roughly coincide.

*The leucocytes.*—In all the cases except case 6 there was well-marked increase of the leucocytes, and even in case 6 there was a very slight increase just before death. The highest leucocytosis in any of the cases occurred in case 8. In this case it rose to 36,875 and the blood was examined on the first day, eighteen hours after the onset of the disease. This was the earliest day on which the blood was examined in any of the cases. Case 2 was examined on the third day after the onset, but the leucocytes on that day were only 16,562, and they rose to a higher point later in the illness. Case 10 is interesting, for although a leucocytosis was known to exist before the onset of pneumonia, when the blood was examined on the fourth day of the disease, the leucocytes had dropped to 14,875, and on the subsequent days to below normal. No doubt the blood disease with which the pneumonia was associated had something to do with this.

In the six crisis cases, the highest point reached by the leucocytes was in cases 1 and 5 on the fourth day of illness and before the crisis; in cases 2, 4 and 7 (the latter before the existence of the otorrhœa and delayed resolution) on the sixth day of the illness. In case 3 the highest leucocytosis occurred during the fall of temperature, thus agreeing with Billings. The mean of the highest leucocytosis in all the crisis cases was 19,362. In cases 2 and 3 there was a rise of the leucocytes during the fall of temperature; but as Billings noticed, this rise was transient, and was soon followed by a fresh fall.

In none of the cases did the leucocytes reach normal at the same time as the temperature, but were always delayed for a day or two; thus, in case 2 the leucocyte curve had reached normal twenty-four hours after the temperature had become normal; in cases 4 and 5, forty-eight hours after, in case 1, three days after,

in case 3, six days after, in cases 7 and 8, both the latter being cases of delayed resolution, fourteen days after.

*The leucocytosis in cases of delayed resolution.*—This is well illustrated in cases 7 and 8. Case 8 is perhaps the most typical of the two, as it was free from the complication of otorrhœa which existed in case 7.

Attention has been already called to the fact that in case 8 the temperature fell by lysis and reached normal on the eleventh day of illness. This seems to have had no effect on the leucocytes, which scarcely altered at all, the figures ranging from 25,000 to 28,000 during the following five days, and then gradually with occasional temporary rises reaching normal on the twenty-fourth day. Throughout the whole three weeks during which the blood was examined the leucocyte curve never attained the height to which it had risen on the first day. The persistent high leucocytosis after the temperature had dropped led to a suspicion of empyema; but, as the leucocyte curve gradually and day by day diminished, this suspicion was not confirmed.

In case 7 otorrhœa started two days after the crisis. Before this, as will be observed, the leucocytes were gradually diminishing in numbers, but the otorrhœa, itself no doubt a considerable cause of the suddenly increased high leucocytosis, was probably also a cause of the delayed resolution in the lungs, as the physical signs persisted for some days after the temperature had reached normal. When the leucocyte curve began to decline, it was observed that the physical signs were clearing up also. As in case 8, the leucocytes did not reach their normal numbers until fourteen days after the temperature. Both these cases of delayed resolution, it will be observed, occurred in children; one in a boy aged 6 and the other in a girl aged 8 years. In case 8 three lobes were affected, in case 7 two lobes. The evidence from these cases goes to confirm the observations of Rieder, Laehr and Billings.

There appeared to be no correspondence between the daily temperature and leucocyte curves in any of the cases. As to the relation of leucocytosis to lung invasion. In case 8, where the left

lower and middle lobes, and the upper right lobe were involved, the average leucocytosis before the temperature dropped was 26,000, whereas in case 7, where both lower lobes were involved, the average leucocytosis was 18,000, and in cases 1, 2, 3 and 4, where only one lobe was involved, the average leucocytosis was also 18,000. Thus with the considerably greater involvement of the lungs in case 8, there was a considerably greater leucocytosis.

*The relation to prognosis in cases of absent leucocytosis.*—As Billings has pointed out, in extremely mild cases of pneumonia there may be no leucocytosis; on the other hand, if in severe cases leucocytosis is absent, an unfavourable prognosis may be given. Of the latter class of cases case 6 is an excellent example. Here with extensive invasion of the whole of one lung the leucocytes were not increased until the day before death, when they rose only a trifle above normal. Notwithstanding that the general condition of the patient was satisfactory on the day the blood was first examined, on account of there being no increase of leucocytes an unfavourable prognosis was given. This prognosis eventually proved correct, as the patient died on the eighth day of illness.

*The leucocytosis in pneumonia complicating typhoid fever.*—Although it has been shown above that in cases of pneumonia where there is no rise of leucocytes, the prognosis is unfavourable, this is not necessarily so in pneumonia complicating typhoid fever. In case 9 on admission patient was found to be suffering from pneumonia and appeared to be in a somewhat serious condition. The blood was examined and as the leucocytes were found to be normal an unfavourable prognosis was given. Two days after admission the true nature of the case was recognised, and a typical Widal's reaction obtained. Throughout the illness the leucocytes never rose above 10,937 per cubic millimetre, and the temperature began to come down on the thirteenth day of illness with a characteristic typhoid remittent chart, and patient was soon convalescent. Pick's case was identical with this, his patient also ultimately recovered.

As to the explanation of these cases, two views appear to be open to us :—

It is a well accepted fact that in cases of typhoid fever the leucocytes do not increase in number and very often a leucopenia exists. Presuming that a marked leucopenia was present in case 9 the pneumonia complicating it, would, if not especially severe, raise the leucocyte count to normal. On the other hand, if we consider that there was no leucopenia and a very mild attack of pneumonia was present at the same time, the leucocyte count would be unaltered; that is, granting that the attack of pneumonia was one of those cases, so mild that no increase of leucocytes was caused.

*Leucocytosis in pneumonia, complicating purpura.*—In case 10, although at the autopsy small patches of broncho-pneumonia were found in the left lung, the physical signs during the early days of the disease pointed to the existence of a right lobar pneumonia. Previous to the onset of the disease the blood had been examined on two separate occasions. One on the twelfth day before the onset, when the leucocytosis was 17,000, and the other on the fifth day before, when the leucocytosis was 16,000. On the fourth day of the disease, and nine days after the last count was made, the blood, on being examined, was found to contain 14,375 leucocytes per cubic millimetre. On the day after they had dropped to 6,875, and on the following day to 2,500. At the time of this leucopenia, which occurred on the fifth day of onset of pneumonia, the patient was in a precarious condition. Following out the suggestion of v. Jaksch, nuclein was given in order to increase the number of leucocytes, with the hope of stimulating the patient to a reaction against the disease. Eighteen hours after the first dose of nuclein had been given, the leucocytes had risen to 9,687, and twenty-four hours later to 26,250, when the nuclein was withdrawn. Patient was decidedly better on the day after the first administration of the drug. The leucocytes continued to remain at, approximately, 20,000, and the physical signs were not found to be clearing up. On the nineteenth day of illness there was a sudden increase of leucocytes to 31,562, and it oscillated about these figures until her death, on the twenty-third day after onset of pneumonia. The sudden increase of leucocytes on the nineteenth day of illness had led to a suspicion of empyema,



and a needle inserted had revealed nothing; but this suspicion, raised by the leucocytosis, was confirmed at the autopsy. Ewing makes the statement that leuchæmia complicating pneumonia reduces the leucocytes; so possibly the existence of purpura in case 10 was the cause of the marked leucopenia present before the administration of nuclein.

*Relation of physical signs to leucocytosis.*—Cases 1, 2, 4 and 5, in all of which the temperature fell by crisis, the leucocyte curve had reached normal a day or two before all physical signs in the lungs had cleared up. In case 3 the physical signs had all disappeared five days before the leucocytes had reached their normal number. In the two cases of delayed resolution the physical signs had cleared up, and the leucocytes had reached normal on the same day. In neither of the cases, 2 and 4, were there abnormal physical signs on admission.

In case 2—

Admitted	...	...	on 2nd day of illness	
Blood examination	...	"	3rd	" 16,562
Physical signs appeared	...	"	5th	"
Leucocytes	...	...	" 6th	" 22,750, highest
Crisis	...	...	" 8th	" [throughout.
Leucocytes normal	...	"	10th	"
Physical signs cleared up	...	"	13th	"

In case 4—

Admitted	...	...	on 3rd day of illness	
Physical signs appear	...	"	6th	"
Leucocytosis	...	...	" 6th	" 18,427
Leucocytes normal	...	"	8th	"
Physical signs cleared up	...	"	9th	"

Therefore, from the above analysis, it is clear that in the majority of cases the leucocytes reach their normal number before all the physical signs have cleared up, generally twenty-four to forty-eight hours before; that the physical signs may clear up before the leucocyte curve has reached normal; and that in cases of delayed resolution the physical signs clear up, and the leucocytes reach normal at much the same time.

## TYPES OF LEUCOCYTES FOUND IN PNEUMONIA.

*The polynuclear cells.*—The mean percentage of polynuclear cells *before the crisis* in cases 1, 2, 3, 4, 7, was 81·4 per cent., the highest in any of the cases being 91·4 per cent. in case 2, and the lowest 73·2 per cent. in case 7. The daily change in percentage numbers before the crisis appeared to be irregular, but varied roughly with the temperature in case 2. *During the crisis* there seemed to be a slight drop in all cases, the mean in cases 1, 2, 4, 5, 7, being 76 per cent.

On the day *after the crisis* there was a still further lowering of the percentage numbers, unless there was a post-critical rise, when they were unaltered. Thus the mean during the day after the crisis in cases 1, 2, 3, 7, was 70 per cent. In case 4 during the post-critical rise it was 75·8 per cent., and in case 5 during the same period it was 74·7 per cent., but on the day after the post-critical rise both dropped to a number corresponding with those which existed on the day after the crisis in the other four cases, namely, to 67·8 per cent. in case 4 and to 70 per cent. in case 5.

*Changes in the percentages of the polynuclear cells from the time the temperature reaches normal until the time when the leucocytes reach normal.*—There appeared to be a gradual diminution in the numbers of the polynuclear cells up to the time when the leucocytes had reached normal, the mean of all the cases when that period was reached being 63 per cent. Except in cases of delayed resolution, it will be noticed on referring to the charts, that the longer the leucocytes take to reach normal after the temperature has arrived at that point the greater the diminution in percentage numbers of the polynuclear cells, and *vice versa*. For instance, in case 2 where the leucocytes reached their normal number on the day after the crisis the polynuclear cells were 80·6 per cent.; whereas in case 3, where the leucocyte curve did not reach normal until six days after the crisis, the polynuclear cells had diminished to 48 per cent.

*The polynuclear cells in cases of delayed resolution.*—During the stage in which resolution was delayed, in neither case 7 nor case 8 were the polynuclears found to remain above normal. The

average in case 7 during this stage being 63 per cent. and case 8 67 per cent., both distinctly normal or below normal; contrary to the observations of Türk, who found them increased above normal during this stage.

*The polynuclear cells after the leucocyte curve has reached normal.*—In all those cases in which the blood was examined after the leucocytes had reached normal, the polynuclear cells were still diminishing in percentage. Thus, in case 7, two days after, the polynuclear cells made up 50 per cent. of all leucocytes; and in case 8, in which blood was examined four days after the leucocyte curve had reached normal, the polynuclear cells made up 45·2 per cent. of all cells, in fact they were exceeded in number by the lymphocytes, which were 47·4 per cent. How long this diminution goes on it is impossible to say from the data in hand.

*The polynuclear cells in severe cases where leucocytosis is absent.*—Rieder, during his examinations, found them increased; Bieganski found them diminished, and Billings normal. Rieder's observations are confirmed by the increase of the polynuclear cells to a marked degree on every day in case 6. On every occasion the polynuclear cells were above 90 per cent., and although their percentage numbers diminished as death drew near their absolute numbers increased.

*The polynuclear cells in pneumonia complicated by typhoid fever.*—In case 9, during the whole of the febrile period, there was a mean percentage of polynuclear cells of 66·8 per cent.; the highest number recorded being 78 per cent. and the lowest 55·8 per cent., the numbers varying irregularly between these figures. As the temperature reached normal the polynuclear cells diminished to 52·4 per cent. The irregularities in numbers otherwise bore no relation to the temperature.

*The polynuclear cells in pneumonia complicating purpura.*—In case 10, on the fourth day of disease, the polynuclear cells were 81·3 per cent., a figure one would naturally expect at that stage of the disease. During the stage of leucopenia they dropped to 50·4 per cent., but soon after the administration of nuclein they rose to 90 per cent., there they remained at numbers ranging between 86 per cent. and 75 per cent., until a few days before

death, when they began to increase once more, and two days before death were 91 per cent. The temperature seemed to bear no relation to their numbers.

*The lymphocytes.*—These cells vary in number with the polynuclear cells. As the polynuclear cells increase their percentages, so the lymphocytes diminish, and when after the leucocyte curve has reached normal and the polynuclear cells are diminishing rapidly, the lymphocytes are found to increase proportionately and sometimes even exceed in numbers the polynuclear cells. Thus, in case 8, where the polynuclears had diminished to 45·2 per cent., and the lymphocytes had risen to 47·4 per cent. Practically all that has been said concerning the polynuclear cells applies to the lymphocytes, if it is remembered that where the polynuclear cells diminish the lymphocytes increase and *vice versa*.

*The large mononuclear cells and transitional cells.*—These two classes of cells are described together, because of the very slight difference which exists between them; the transitional cells being a slightly older cell than the large mononuclear.

- Looking at the tables and comparing the numbers of either variety of cell separately, or the two together, the fluctuations in their numbers seem to bear no well-defined relation either to the temperature or leucocyte curves. They are certainly not diminished, nor are they increased with regularity during the height of the fever, or during a large increase of the leucocytes; but perhaps, after the crisis, an increase in their number may be detected during the first few days. Thus in case 2, two days after the crisis, the combined percentages were 4·2 per cent. In case 4, two days after the post-critical rise, they were 8·8 per cent. In case 7, two days after the crisis they were 14·2 per cent. (in this last case the figures were above normal throughout). In case 3 there was no marked reaction until five days after the crisis, when they rose to 7·6 per cent. It will be observed in this last case that the leucocyte curve was delayed in coming down to normal.

Previous to the crisis no regularity seems to exist, except in case 2, where there was a gradual diminution of the combined

figures towards the crisis. In case 7 the reverse existed, a gradual increase towards the crisis being noticed.

In all, except in very few instances, the transitional cells are always in excess of the large mononuclear cells.

No relation appears to exist between the numbers and the amount of lung involved.

*Changes in the eosinophile cells.*—The course of the eosinophile cells is seen very plainly on the charts. In none of the cases were they found during the febrile period. In cases 1 and 5 they appeared during the fall of temperature by crisis, and in all other cases they were found immediately after the temperature had reached normal. In case 7 they were present in normal numbers during nearly the whole of the stage of delayed resolution, even when the leucocytosis was at 36,000 per cubic millimetre, and in case 8, where the temperature fell by lysis, they began to appear during the fall of temperature, and were frequently found during the stage of delayed resolution, though not with the same regularity as in case 7.

In case 9 (typhoid and pneumonia) they only appeared during the period of fall of temperature.

In case 10 (purpura and pneumonia) they were very scanty, though not entirely absent throughout the disease.

In the case in which there was no increase of leucocytes, which proved fatal (case 6), eosinophiles appeared in small numbers on the day preceding death, not having been found in the films on either of the previous days, although on one of these days 1,000 cells had been counted, and the whole surfaces of the films well hunted in addition for their presence.

*The presence of myelocytes in pneumonia.*—These cells, although not present in normal blood, have been found in most of the cases of pneumonia examined, at some time or other during the disease. Case 2 was the only one of the series in which they were entirely absent. They were rarely present during the febrile period, except on the day previous to the crisis, and in those cases where complications existed. They were found in cases 3, 4 and 7, on the day immediately preceding the crisis, in

cases 3, 5, 7 and 8 on the day after the temperature had reached normal, in case 1 on the second day after, in case 4 on the third day after, and in case 9 two days after the temperature had reached normal. In case 7 they were found on two occasions during the febrile period and were also present in small numbers during the stage of delayed resolution but never above 0·8 per cent. In case 8, likewise, they were present in small numbers on two occasions during the febrile period, there was an increase to 1·4 per cent. immediately after the temperature had fallen, and they appeared on frequent occasions during the period of delayed resolution. In case 9, in addition to their appearance after the temperature had reached normal, they were also found during the febrile period. Cases 3 and 10 both showed comparatively large numbers in the blood. In case 3, on the sixth day after the crisis, they had risen to 7 per cent., and had been gradually increasing in numbers from the fall of temperature up to that day. A special curve for the myelocytes has been drawn on the chart.

In case 10 it is impossible to say how far the purpura may have been responsible for the large numbers of myelocytes, but I may say that there was no suggestion of leukæmia in the case, as the blood was absolutely unlike that of a leukæmic patient, both in number of eosinophiles and in the comparatively small increase in number of leucocytes, as well as in the preponderating polynuclear character of the leucocytosis, the size of the myelocytes found, and in the incomparably smaller absolute number of myelocytes present. It will be seen by the charts that the first appearance of the myelocytes dated from the period of leucopenia on the sixth day of illness, ten days later they had reached a total of 10·1 per cent., or 2,114 absolute, and gradually dropped again before death. In Türk's case 11·9 per cent. was the highest number reached. About 70 per cent. of the myelocytes found in the above cases were the size of a polynuclear cell, the remaining 30 per cent. being of large size, approaching and sometimes even exceeding in size that of a large mononuclear cell.

*The presence of mast. cells in pneumonia.*—From an examination of the tables the presence of mast cells seems to be associated with the eosinophile cells. In the whole series of cases they were only found on two occasions unassociated with eosinophiles, namely, once in case 8, and once in case 10. In all other cases eosinophiles were always present when these cells appeared. In six cases they were found during one of the three days immediately following the crisis, but in case 3 they did not appear until the fourth day after the crisis. They were only found occasionally during the stage of delayed resolution, and eosinophiles were always present at the same time. Case 6 was the only one of the series in which no mast cells were found in any of the blood films, although the films were searched thoroughly after counting had been discontinued.

GENERAL CONCLUSIONS CONCERNING THE CONDITION OF THE  
BLOOD IN PNEUMONIA.

*The red corpuscles.*—

- (1) During the febrile period the red corpuscles are practically normal.
- (2) During the crisis in the majority of cases they diminish in numbers.
- (3) After the crisis they may rise again to normal during the next few days, or may remain down for some days.
- (4) The chief value of the estimation of the red corpuscles when the leucocytes are being examined is to prove whether the blood is concentrated or not.
- (5) Nucleated red corpuscles are rarely present except in cases of delayed resolution, and then only in small numbers. They may be also present in other complications.

*The hæmoglobin.*—

The course of the hæmoglobin is roughly that of the red corpuscles.

*The leucocytes.*—

- (1) In the large majority of cases of pneumonia there is a marked increase of leucocytes. Cases occur in which there is no increase, and generally prove fatal. In cases of pneumonia complicating typhoid fever and purpura there may be no increase of leucocytes, and the absence of leucocytosis in pneumonia complicating typhoid does not necessarily mean an unfavourable prognosis.
- (2) There is no evidence to show at which stage of the disease the leucocytes reach their highest point; but there are reasons for thinking that this point is reached soon after the onset of the disease.
- (3) There may be a slight rise of leucocytes during the period of fall of temperature.
- (4) The temperature curve always reaches normal before the leucocyte curve; the latter may take from one to three days longer to reach the normal, but in some cases it may be delayed for some days, even though the lungs are resolving. In cases of delayed resolution the leucocytes remain elevated after the temperature has reached normal, and may not fall until the lapse of some weeks.
- (5) There is no correspondence between the temperature and leucocyte curves.
- (6) There appears to be a rough relation between the amount of leucocytosis and lung invasion.
- (7) In cases of severe uncomplicated pneumonia, with no increase of leucocytes the prognosis is unfavourable.
- (8) In cases of typhoid fever complicated by pneumonia there may be no leucocytosis, but the prognosis is not necessarily fatal.
- (9) In cases of pneumonia complicating purpura there may be a diminution of leucocytes below normal even though a leucocytosis existed before the onset of pneumonia. In such a condition the administration of nuclein may improve the general state of the



patient by increasing the leucocytosis, and a sudden rise of leucocytes during a prolonged leucocytosis may indicate the presence of empyema.

- (10) In the majority of cases the leucocytes reach their normal numbers before the disappearance of all abnormal physical signs. In cases of delayed resolution the abnormal signs clear up and the leucocytes reach normal about the same time.

#### TYPES OF LEUCOCYTES FOUND IN PNEUMONIA.

##### *The polynuclear cells.*—

- (1) These cells are increased above normal during the febrile period, the daily change in their percentage during this time being irregular.
- (2) During the crisis there is a slight drop in numbers, in all cases; and on the day after the crisis there is a farther lowering of their percentage numbers, unless there happen to be a post-critical rise of temperature, when they remain unaltered until the day following this fresh rise, when they behave as on the day after a crisis.
- (3) In cases of delayed resolution, when the temperature has reached normal and the leucocytes remain excessive, the polynuclear cells are not increased above normal, but are generally either normal or subnormal.
- (4) After the leucocyte and temperature curves have both reached normal, the polynuclear cells still go on diminishing, and may even diminish to such an extent as to be exceeded in numbers by the lymphocytes.
- (5) In some severe cases where leucocytosis is absent the polynuclear cells considerably increase above normal, but they may be normal or subnormal in numbers (Bieganski and Billings).
- (6) In cases of pneumonia complicating typhoid fever, where no increase of leucocytes exists, the relative percentages of the polynuclear cells is normal; but may become subnormal as the temperature falls.

- (7) In cases of pneumonia, complicating purpura, if a leucopenia exist, the polynuclear cells may fall considerably below normal.

*The lymphocytes.—*

The percentage numbers of these cells vary inversely as the polynuclears; that is to say, as the polynuclear cells increase the lymphocytes diminish, and *vice versa*. In the latter case the lymphocytes sometimes exceed the polynuclears in number.

*The large mononuclear and transitional cells.—*

- (1) During the whole course of the disease these cells follow an irregular course; sometimes increasing in numbers, sometimes diminishing. These changes bear no relation to the temperature, the extent of exudate, or the leucocytosis. In some cases there may be a slight increase in their numbers within the first few days after the crisis or post-critical rise of temperature.
- (2) The transitional cells nearly always exceed the large mononuclear cells in number.

*The eosinophile cells.—*

- (1) Are rarely if ever present during the febrile period.
- (2) Sometimes begin to appear during the crisis, but more commonly immediately after the crisis.
- (3) Are generally present during a delayed resolution, even though the leucocyte curve remains elevated.
- (4) In cases of pneumonia complicating typhoid fever they only appear during the fall of temperature.
- (5) In cases of pneumonia complicating purpura they are very scanty, though not entirely absent throughout the disease.
- (6) They are occasionally present in those fatal cases where leucocytosis is wanting.

*Myelocytes.*—

- (1) In the majority of cases they are present during some stage of the disease.
- (2) Frequently appear on the day preceding the crisis and during the crisis.
- (3) Are nearly always present on the day after the crisis and continue to appear during the subsequent days for a doubtful length of time.
- (4) In some cases they may be present in comparatively large numbers after the temperature has fallen.
- (5) During the stage of delayed resolution they appear in small numbers.
- (6) Are often present when eosinophiles are absent.

*Mast. cells.*—

- (1) Rarely appear unassociated with eosinophiles.
- (2) Frequently appear during one of the first three days following the crisis.
- (3) Are completely absent in severe uncomplicated cases where there is no increase of leucocytes.

## PART II.

### RECORDS OF SOME BLOOD EXAMINATIONS DURING THE HEALING OF WOUNDS.

In the following account of ten cases in which the blood was examined during the healing of wounds, the chief object has been to ascertain whether the different types of cells altered in their relative proportions to one another, or whether they retained their normal numbers after an operation in which the wound healed by first intention. Secondly, to examine the changes in numbers of leucocytes which occurred after an operation. (a) In which the wound healed by primary union. (b) In which the wound healed by granulation. As to the work done by others on this subject, as far as can be ascertained either no work has been done or it has not been recorded in such a manner as to obtain

publicity. Of this I am certain, that there is no literature on the subject in the English language, and all the principal foreign writers on hæmatological matters, namely, Ehrlich, Hayem, Grawitz and Limbeck, make no mention of the normal condition of the blood during the healing of wounds. Also I have been unable to find references to the subject in any of the indices or digests which exist. Cabot (Guide to Clin. Exam. of Blood, 1900) deplores the absence of work on this subject in the following words (describing a wound he had to deal with): "The wound was healthy, freely discharging, and had healed satisfactorily at the time of the last count recorded, and yet during its healing the leucocytes had remained more or less above normal for a month. Whether all wounds follow this course I do not know. It is an important point which needs working out, namely: What is the normal behaviour of the blood during the healing of granulating wounds? If this were known we might get valuable information as to whether a wound is doing well or not, by means of the blood count, which if septic would probably behave differently from its wont in wounds which do well. As it is, all these questions are not answerable. It is hoped that surgeons will investigate them."

Ewing, whose text-book (Clin. Path. of the Blood, 1901) on general hæmatology is, I believe, the latest and most complete extant, does not mention the condition of the blood during the normal healing of wounds, but describes the diminution of red corpuscles and hæmoglobin, and increase of leucocytes which exist in septic fever. He also says: "Regarding the more minute factors determining the grade of leucocytosis in septic wounds little is known."

Whether any literature does exist and whether the blood has been examined during the healing of wounds by other observers I have been unable to determine, consequently I shall be happy to be informed of their existence if I am wrong.

The methods of examination, technique, etc., are the same as those described in Part I. of this paper. The charts and tables are drawn up on the same lines.

CASE 11. *Extensive varicose veins both legs. Primary union.*—H. P., æt. 19, admitted under the care of Mr. Howse, November 27th, for varicose veins in both legs. History of case.—Veins first appeared six years before admission, and had been gradually increasing in size since that time. He was troubled with constant aching in them, especially at night. *Condition on admission.*—Patient was a strong muscular lad. There was a bunch of enlarged veins at back of right leg, also another extending from inner side of ankle to inner side of right knee. There was also a large varicose vein running from saphenous opening along inner side of left thigh to knee and from knee to inner side of ankle. Operation.—November 30th. A long incision was made over the veins in the left thigh extending from saphenous opening to just above knee. Veins were removed and collateral branches ligatured. Another incision was made just below left knee to about an inch above the ankle. The vein was treated in the same manner. From the right leg veins were removed also, but the incisions were not quite so extensive. All the wounds were sewn up with continuous gut sutures. Total wound length nearly twenty-five inches. December 4th. Patient said that the wounds pained him slightly, but they looked perfectly healthy when he was dressed. December 9th. All wounds had healed up by primary union and stitches were taken out.

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CASE 12. *Varicose veins in both legs removed. Primary union.*—J. H., æt. 32, admitted under the care of Mr. Howse, October 17th, with extensive varicose veins in right leg, and a few in the left leg. Previous illnesses.—Has had malaria in 1887, in Pietermaritzberg. In 1890 he had a slow, continuing fever, with ague, in Mauritius. History of case.—Patient first noticed veins in right leg to be swollen about three years ago. They, however, were not of large size till about eight or ten months ago, when they became enlarged to almost their present size. During the last four or five months he has had pain in them while walking. *Condition on admission.*—Patient is a strong, healthy and active man. The varicose veins in the right leg are situated chiefly on the back and inner side of the leg. When the patient lies down they are not so noticeable; but when he stands up their tortuous outlines become very prominent. There appears to be no thrombosis. A few veins exist in the left leg also. Operation.—October 23rd, A.C.E. was administered. Three large incisions were made over the veins in the right leg. The veins were ligatured, and as large a part as possible was excised between the ligatures; the wounds were then sewn up with gut. The few veins in the left leg were treated in the same way. The total linear length of the wounds amounted to from twelve to fifteen inches. The wounds were dressed, and both legs were placed on back splints. October 28th. Wounds redressed and are looking well. November 1st. Stitches were taken out to-day. Wounds have healed by first intention, and a sealed dressing was applied to each.

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CASE 13. *Osteo-arthritis of knee. Joint opened. Redundant portions of mucous membrane removed.*—T. C., æt. 33, admitted under the care of Mr. Howse, October 10th, for pain in right knee-joint, worse at night. History of case.—Patient had been attended one month previously for misplaced semilunar cartilage, and his right knee had been put up in a Bavarian splint, but

the pain in the knee did not improve and he was taken into the hospital for further examination. *Condition on admission.*—The right knee is considerably swollen, especially above and on the right side of the patella. The swelling fluctuates, and a marked patellar tap can be obtained. Full extension of the leg on the thigh can be obtained, also flexion, but the latter only to an angle of thirty degrees. No pain on movement or forcibly increasing flexion. The thigh muscles on the right side are somewhat wasted. On the outer side of the knee about one inch above the lower end of the condyle is a small hard body about three-quarters of an inch long and half an inch broad, freely movable backwards and forwards, but not upwards and downwards. Pressure over this is painful. Just on inner side of patella is a similar body one inch long and a quarter of an inch broad extending below level of patella, with its lower end not very definite. This is also very painful on pressure. Just as he is dropping off to sleep he is awakened by sharp pain in the knee. It was decided to operate on the knee-joint. Operation.—October 15th, Patient was put under A.C.E. and ether. Two vertical incisions were made one on either side of the patella. A small quantity of serous fluid escaped as the joint was opened. Several small bodies attached to the synovial membrane were removed from either side of the joint, the largest of these being the one which could be felt under the skin previous to the operation. It was obviously a case of osteo-arthritis of the joint. The joint after being well syringed out with a lotion of perchloride of mercury, was closed with silkworm gut sutures and the skin sutured with five gut sutures on each side. An ice-bag was applied to the knee after the patient was removed back to bed as there had been considerable oozing. October 17th, wound redressed. October 18th and 19th, wound dressed. October 20th, ice-bag discontinued. October 21st, wound redressed. October 25th, stitches taken out, wound has healed by first intention, there being no trace of inflammation about the wounds. October 28th, wound redressed.

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CASE 14. *Appendix removed, primary union.*—R. B., æt. 42, admitted under the care of Mr. Howse, October 30th, with a view to removal of appendix. Previous illnesses.—Patient had pleurisy thirty years ago and small-pox fifteen years ago. History of case.—He had an attack of appendicitis about five months ago, another one three months ago, and one just before admission. *Condition on admission.*—Patient is an unhealthy looking man and rather inclined to worry himself about trifles. Pulse 74, temperature 99.4, respiration 25. A thickening can be felt in the region of the appendix and there is slight tenderness over this spot. All pain had disappeared by November 2nd, but it was decided to wait for some days before operating. Operation.—November 12. An incision six inches in length was made over the region of the appendix. The appendix was found to be bound down by adhesions. These were divided, the appendix drawn out of the wound, ligatured, and the peritoneum stitched over the stump. The abdominal wound was stitched up with salmon gut. November 20th. Wound redressed and looked quite healthy. It had healed up by primary intention. Stitches removed. November 22nd. Wound redressed and sealed dressing applied.

CASE 15. *Strangulated Hernia. Primary Union.*—J. H., æt. 18, admitted under the care of Mr. Howse, October 18th, for swelling in the right inguinal region, pain in the stomach and sickness. History of case.—Patient, who is a strong, healthy young man, had noticed a swelling in the right inguinal region some months before admission, but did not suffer any inconvenience from it until the morning of admission, when at 9.30 a.m., while at his work, he was seized with great pain in the abdomen. A doctor was sent for, who ordered his removal to the hospital. *Condition on admission.*—October 18th, 2 p.m. Soon after admission he vomited up some brownish green fluid, and complained of great pain in the epigastric region. A tense swelling was found in the right inguinal region, which with other signs, pointed to the existence of a strangulated hernia. The blood was examined soon after admission, and an account of the results are given in the tables. At 2.45 p.m. Patient was put under an anæsthetic, and an incision was made over the swelling. The hernial sac was reached and opened up, when a gush of serum-like fluid took place from it. The intestines were blue and congested, but there was no fecal odour. The hernia was reduced, and the abdominal ring closed with silk sutures. There was very little bleeding during the operation. October 21st. Wound was dressed and looked quite healthy. October 28th. Stitches were taken out to-day. The wound had healed by first intention.

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CASE 16. *Tuberculous glands in neck removed. Slight suppuration.*—R. M., æt. 26, admitted under the care of Mr. Howse, November 21st, for enlarged glands in neck. History of case.—Patient had been in the hospital during the previous September and on September 9th a mass of caseous glands had been removed from the right side of neck. The wound healed up rapidly and he was discharged from the hospital on October 14th. At the time of his operation all the glands could not be removed, so he was told to come up on a future date for a further operation. After the last operation patient had an attack of ague. Since his discharge the glands remaining on right side of neck had increased in size. He also complained of some loss of power in his right arm. *Condition on admission.*—Pulse 60, temperature 96.4°, respiration 20. Patient was a rather pale individual with no great muscular development. There were two swellings on right side of neck, one just below angle of jaw. The other was just beneath the old scar on the surface of the sterno-mastoid. Both swellings were about one and a half inches in diameter and did not fluctuate. Operation.—An incision four inches long was made over the glands and they were dissected out, but other glands were discovered adherent to the carotid sheath. An attempt was made to remove these by making another incision at right angles to the former and behind it joining it at its centre, but then only a limited amount of gland tissue could be removed and a large part of it had to be left. The wound was closed with a continuous gut suture. There was considerable hæmorrhage during the operation. December 5th. Wound dressed; it was quite sweet and clean. Temperature rose during the evening and he had a typical ague attack with shivering, etc. December 9th. Wound redressed. The upper part of it had closed, but the hinder part was gaping and there was a little discharge. December 11th. Hinder part of wound was suppurating. A portion of the excised glands showed on examination a typical tubercular

structure. December 13th. Stitches taken out of the anterior part of wound which had healed up. December 16th. Patient was discharged from hospital. The posterior portion of wound was still discharging. The part of the incision first made had healed up by primary union.

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CASE 17. *Tuberculous glands in neck (breaking down) removed.*—A. F., æt. 8, admitted under the care of Mr. Howse, November 21st, for tuberculous glands in neck. History of case.—Swellings first appeared in neck a month before admission. Since that time they had been gradually increasing in size. *Condition on admission.*—Temperature 98·6°, pulse 76, respiration 30. There were three small glands, one on the right side, about three inches below the ear and posterior to the sterno-mastoid. This gland was found to fluctuate on palpation. The second was just over the inner extremity of the right clavicle, and the skin over it was inflamed, but no fluctuation could be detected. The third was about one inch above the inner extremity of the left clavicle. This also did not fluctuate. Progress.—On November 26th, the swelling over the right clavicle had become considerably larger, and appeared to be breaking down, as fluctuation could be obtained. The gland over the left clavicle had diminished in size. Operation.—November 28th, A.C.E. was administered. An incision was made into the swelling over the right clavicle, and about half a teacupful of pus was evacuated. The same was done to the swelling below the ear, about a tablespoonful of pus being removed. The wounds were washed with perchloride of mercury, and iodoform emulsion was poured in, the cavities being packed with iodoform gauze. December 3rd. Wounds redressed. They appeared to be quite healthy, and there was no discharge. The wounds were drawn together with sutures as far as possible. December 7th. Wounds redressed, and were doing well. They were very superficial, and were healing by second intention.

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CASE 18. *Hernia. Radical cure. Healing by second intention chiefly.*—G. W. E., æt. 25, admitted under the care of Mr. Howse, November 28th, for left inguinal hernia. History of case.—Patient first noticed swelling twelve years before admission. It has become larger during the last two years, but he has never had any difficulty in reducing it. *Condition on admission.*—Patient was a slightly built man somewhat pale in face, but appeared to be otherwise healthy. The tumour in the groin had all the characteristics of an inguinal hernia. Operation.—December 5th. An incision about four inches long was made over the hernia, its centre being over the external abdominal ring. The sac was opened and found to consist chiefly of omentum with a small portion of intestine. The omentum was ligatured and cut off and the intestine was reduced. The abdominal parietes were brought together with silk sutures and the skin was closed with gut sutures. December 9th. Patient was redressed. About an ounce of fluid blood exuded between two sutures and the operation dressing had been soaked through with blood. No pus. The wound was being dressed every day. December 12th. There was a slight serous discharge from surface of wound and some broken down blood-clot was squeezed out from centre of wound. No pus seen. December 14th. Wound had healed at both ends but for a space of two and a half inches there was a discharge of serum and dark viscous blood.



December 17th. Centre of wound was granulating. Both ends had healed. No pus had been detected and there was only a little serous discharge. It was thought that the discharge of dark viscous blood from the wound might be the precursor of pus, but no pus was ever detected and centre of wound slowly healed by granulation in about three weeks after operation.

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CASE 19. *Strangulated hernia. Operation. Suppuration.*—F. W. K., æt. 42, admitted under the care of Mr. Howse, November 19th, with strangulated right inguinal hernia. History of case.—Patient has had a hernia on his left side for many years. Eight years ago, while lifting a piano, he produced a hernia on his right side. He has worn a double truss since that time. On November 18th, while lifting some heavy furniture, the hernia on his right side came down and he was unable to return it. He was in great pain and a doctor ordered the application of an ice-bag to it. On November 19th the doctor again saw him and attempted to reduce the hernia; being unable to do so he was sent to the hospital. *Condition on admission.*—Patient is a strong, healthy looking man. He complains of great pain across the stomach. The bowels have not been open since the hernia came down, and he has been sick two or three times, the last time being at 6 a.m. on the day of admission. On no occasion was the vomiting fecal. He has a very large hernia, extending from the internal abdominal ring to nearly the bottom of the scrotum. It is about the size of two fists, very tense, entirely irreducible, dull on percussion and gives a scarcely perceptible impulse on coughing. The testicle lies below the hernia and the latter is very tender to the touch. The blood was examined immediately before the operation. *Operation.*—A large incision was made, from a little above the internal abdominal ring nearly to the bottom of the scrotum. The sac was exposed and found to contain a huge mass of omentum, constricted at both external and internal abdominal rings. A small amount of bowel was also present, which was slightly congested. The bowel was reduced and the omentum ligatured and cut off. Part of the sac was removed after being ligatured and part which was extremely adherent left *in situ*. The abdominal parietes were drawn together with silk and the skin closed with a continuous gut suture. November 20th. Patient's bowels were open for the first time since the operation. November 21st. Temperature has risen to 103.5°. Wound dressed during the evening looked satisfactory. November 25th. Wound redressed. There was brawniness and œdema above the wound towards the iliac fossa. Great tenderness on pressure over this, and during the pressure about two teaspoonfuls of pus escaped from the lower part of the wound where union had failed. November 26th. Wound redressed. About two teaspoonfuls of pus again squeezed out of lower part of wound. The brawniness and œdema above upper part of wound had increased. Upper part of wound was reopened with dressing forceps. November 28th. Brawniness and œdema had diminished. Discharge less. The wound was open at both ends and had healed in the centre. There was an underground connection between the open parts. December 1st. Wound looked much better. Discharge of pus less. December 3rd. Wounds being dressed daily. Discharge of pus much less. December 4th. The remainder of the stitches were taken out, those around the open ends having been previously removed

to allow of free drainage. There was a small wound at lower part of incision from which pus could still be squeezed, and a larger wound above with a sinus extending upwards towards the anterior superior spine. December 7th. As he was getting rather anæmic iron and arsenic was prescribed. (This did not appear to affect the blood count.) A sample of pus from the wound was examined bacteriologically and found to contain a bacillus not unlike the Klebs-Löffler bacillus but non-pathogenic to rabbits. A few cocci were also present. December 12th. Hot boracic fomentations were first applied as a dressing to the wound at 2 p.m. on this day. December 24th. The wound slowly granulated. The lower part had been allowed to heal, but the upper part was still open and discharging a little. A sinus extended downwards from this for about one inch.

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CASE 20. *Severe burns.*—E. W., æt. 10, admitted under the care of Mr. Howse, November 17th, for severe burns. History of case.—Patient was playing with the fire in her bedroom, when her clothes caught alight. Her stepfather hearing her screams, rushed in, and managed to stifle the flames. The accident took place at 6.30 p.m. on November 17th. *Condition on admission.*—The chief parts burnt were :—The whole of the right arm (burn of third degree), the upper part of the anterior surface of right thigh, and lower part of right half of anterior abdominal wall (burns of second degree). There was also an erythematous patch on left half of anterior abdominal wall (first degree). Patient was brought to the hospital at 7 p.m. She was very restless; but her pulse was fairly good, and she showed no signs of shock. The burns were treated with boracic fomentations. Progress.—November 18th. At 2.30 a.m. patient showed signs of collapse, the pulse being hardly perceptible at the wrist. At 4.30 a.m. two pints and a-half of saline solution were infused into the veins. This had a very stimulating effect, but brandy and strychnine had also to be administered freely. November 19th. The temperature, which during the previous days had been subnormal, rose to 103°. She was having frequent saline infusions into the veins, together with strychnine and brandy to counteract any collapse. November 26th. The skin over the wounds of the second degree came off on the dressings, and most of the wounds were discharging pus, a large amount being found on the dressings. There had been a little sloughing of the tissues on upper part of right arm, an area about two inches square being so affected. Boracic fomentations were replaced by hot boracic baths. December 2nd. Sloughs had separated from the right arm and all the wounds had started to granulate. From this time onwards progress was slow, but granulation took place steadily, so that on December 15th zinc ointment could be used as a dressing.

Among these ten cases, five healed by primary union, two suppurated and then healed up by granulation, while of the other three, in one, although there was no actual suppuration, the wound was prevented from healing by the presence of blood beneath the skin, which prevented union of the tissues, and the wound eventually granulated; in another of the three there was

slight superficial suppuration of a small portion of the wound, and in a third case an abscess existed before operation, after which it healed by union of the granulations.

*Wounds healing by primary union.*—In cases 11, 12, 13, 14 and 15, every wound healed by first intention. Except in case 13, the blood was examined in every case previous to operation, in order to obtain an idea of the normal condition of the blood in each patient. It will be observed that in all of these five cases the original disease from which the patient was suffering was not such as to alter the blood at all from the normal, except in case 15, in which there was a leucocytosis to start with. In case 14 the appendicitis had subsided some time previous to the operation. Due precautions were taken to avoid digestion-leucocytosis when procuring the blood.

*The red corpuscles.*—Many factors tend to alter the relative numbers of the red corpuscles, *e.g.*, effects of starvation, vomiting, hæmorrhage, diarrhœa, etc., but in the above five cases these complications have not been frequent, and where they were present have been mentioned in the reports of the cases. As to the effects of starvation, every patient suffered slightly from its effects, as food was necessarily withheld for some hours before each operation, and also for a short time subsequent to it. With regard to the effects of hæmorrhage, in none of the five cases was this excessive; but in case 13 there was a little oozing after the dressings had been applied. Case 15 had not taken any food since the onset of the strangulation, and in addition to this had vomited on one or two occasions previous to admission, but had not done so after the operation. Case 13 was not examined until forty-eight hours after the operation, consequently the effects on the red corpuscles was not noted. A good deal of irregularity in numbers, both of the red corpuscles and the hæmoglobin, is noticed in every case. Cases 12 and 14 were the only ones of the five to show a marked increase in the numbers of the red corpuscles immediately after the operation, the increase in case 12 being 650,000, and in case 14 being 1,250,000, but in both cases there was a diminution of the hæmoglobin. In case 11 there was a slight diminution of the red

corpuscles during the two days after the operation, the hæmoglobin remaining unaltered, but on the third day there was an increase of 1,000,000 red corpuscles, and an increase of 4 per cent. in the hæmoglobin. In case 15 the concentration of the blood produced by the vomiting, etc., previous to the operation caused an apparent diminution in the numbers of the red corpuscles on the day after the operation, but on the second day there was a considerable increase both in the red corpuscles and in the hæmoglobin. Case 13 was not examined immediately after the operation, but the figures were above normal when the blood was examined forty-eight hours later.

From these figures it is impossible to draw universal deductions, but there certainly appears to be an increase of red corpuscles soon after the operation, and the hæmoglobin changes are uncertain.

*The leucocytes.*—Cases 11 and 14 both show a slight increase in the leucocytes during the two days following the operation. In case 12 there is also an increase but not above what is considered normal. In case 13 on the third day there was still a slight increase of leucocytes, which fell on the fourth day but was again above normal on the fifth and sixth days. Case 15 who had a leucocytosis of 26,000 on admission, showed a drop of the leucocytes to 13,000 on the day after the operation gradually decreasing to normal on the fourth day. We see, therefore, that after operation there may be a slight leucocytosis, in some cases this increase not being above normal, that in all cases this temporary leucocytosis gradually disappears in two or three days if the wound is going to heal by primary union.

*The relation of the leucocytes to the temperature.*—In all these five cases except in case 13 the temperature was under 100° on the day after the operation. In cases 11, 14 and 15 the temperature varied between 99° and 99·6° and the leucocytes between 12,000 and 14,000. In case 12 the temperature was subnormal and the leucocytes 8,750. In case 13 the blood was not examined until forty-eight hours after the operation, when the temperature was 100·6° and the leucocytes 10,312. On the day after, the temperature was still slightly elevated and the leucocytes

had risen to 12,187; on this account the wound was examined but found to be quite satisfactory. On the fourth day both leucocytes and temperature had fallen to normal. Therefore, a slight elevation of both temperature and leucocyte curves during the two or three days immediately following the operation is not an indication that the wound is not healing satisfactorily. During the slight leucocytosis which generally follows an operation, the temperature, as a rule, is scarcely increased above normal limits in cases where healing by first intention is in progress.

#### CHANGES IN NUMBERS OF THE DIFFERENT TYPES OF CELLS IN CASES OF PRIMARY UNION.

*The Polynuclear cells, after operations.*—In cases 11 and 15 on the day after the operation the polynuclear cells were less in numbers than previous to it, notwithstanding the existence of a slight leucocytosis. In case 12, where both temperature and leucocytes were normal, there was an increase of these cells on the day after the operation, but they began to diminish on the day following. In case 13, at the time when the temperature and leucocyte curves were oscillating, and there was a suspicion that the wound might be the cause, the polynuclear cells were never increased above normal, in fact they were below normal. In case 14 there was a considerable increase of these cells during the day after the operation, and a leucocytosis of 14,062 existed, but they had dropped to normal on the following day and continued to diminish during the healing of the wound.

In all the cases the highest number reached on the day after the operation by the polynuclear cells was 84·8 per cent. in case 14, the lowest number being 73·6 per cent. in case 11.

*The Polynuclear cells during the later stages of healing by first intention.* - From the second to the third day after operation all the charts show a gradual diminution of the polynuclear cells, which appear to reach their minimum numbers between the sixth and fourteenth days. Thus :—

Case 11	reached minimum	6th day	60·6	per cent.
Case 12	"	"	11th day	54·6 "
Case 13	"	"	14th day	55 "
Case 14	"	"	9th day	54 "
Case 15	"	"	7th day	49·2 "

*The lymphocytes during healing by first intention.*—Although these cells follow an opposite course to that of the polynuclear cells, namely, as the polynuclear cells diminish so the lymphocytes increase in numbers, in certain cases they seem to reach their maximum before the polynuclears have diminished to their minimum, in other cases they are delayed until after this has taken place. This will be best seen by comparing the foregoing table of minimum numbers of polynuclear cells with the following:—

Case 11	maximum of lymphocytes reached	8th day	27	per cent.
Case 12	"	"	11th "	39·8 "
Case 13	"	"	7th "	23·2 "
Case 14	"	"	9th "	37 3 "
Case 15	"	"	11th "	38 "
				average 33 "

It will be seen that cases 12 and 14 were the only ones of the series in which the lymphocytes had reached their maximum and the polynuclears their minimum on the same day. In cases 11 and 15 the polynuclears were previous, in case 13 the lymphocytes were in advance. It will be remembered that cases 11 and 15 were the only cases in which the polynuclears were diminished on the day following the operation.

After operation, therefore, in cases where healing by primary union is going to take place, the lymphocytes appear to increase as the polynuclear cells diminish. They progressively increase during the healing of the wound, reaching their maximum before, after, or on the same day as the polynuclear cells reach their minimum. In cases where the polynuclear cells have diminished on the day after the operation, these cells are more likely to reach their minimum before the lymphocytes reach their maximum.

*The large Mononuclear and Transitional Cells during healing by first intention.*—These two classes of cells being closely associated are described together.

In all cases there appears to be an increase of these cells a few days after the operation. Although there is no immediate effect on their numbers at the time of the operation, soon after, they begin to increase, reaching a maximum either previously or at the same time as the polynuclears.

Table showing dates on which large mononuclear and transitional cells reached their maximum numbers.

Case 11	reached maximum	5th day	10·2	per cent.	Increase chiefly transitional
Case 12	"	"	4th day	11·3	" " " "
Case 13	"	"	14th day	18·3	" Both varieties increased
Case 14	"	"	6th day	7·0	" Transitional only increased
Case 15	"	"	7th day	12	" Both varieties increased

*The Eosinophile cells in cases of healing by first intention.*—The increase of eosinophile cells above normal a few days after operation is perhaps the most noticeable feature in the charts. Thus :—

Case 11	eosinophiles	6	per cent.	on 5th day	after operation
Case 12	"	4·8	"	"	7th " "
Case 13	"	5·2	"	"	6th " "
Case 14	"	5·2	"	"	12th " "
Case 15	"	7·0	"	"	5th " "

Cases 14 and 15 were the only ones of the series in which eosinophiles were not found on the day after the operation, although in both cases they had been found previous to the operation. In all except case 13 their minimum numbers were present on the day after the operation. In case 13 the blood was not examined on the day after the operation.

It will be seen by the above table that in four out of the five cases the maximum was reached between the fifth and seventh days, but in case 14 they were delayed until the twelfth day.

In case 17, which was practically a case of primary union, the same increases of eosinophiles was observed.

Certainly, therefore, in a large number of cases, if not in every case of healing by primary union, there is a reaction of the eosinophiles above normal within a few days of the performance of the operation.

*Myelocytes, nucleated red cells, and mast. cells, in cases healing by primary union.*—Myelocytes were only found on one occasion, namely, in Case 13 on the seventh day.

Nucleated red cells found on one occasion in case 13 on third day, and on one occasion in case 15 on fourteenth day.

Mast. cells were found in every case at some period or other.

In cases 11 and 14, they appeared on the day following the operation, in case 11 being especially plentiful.

On only one occasion were they found when eosinophiles were not discovered, namely, in case 14 on the first day after the operation. Neither of the three last described types of cells appear to bear any relation to the condition of the wounds.

Of the remaining five cases, cases 16 and 17 were operations on tubercular cervical glands. In case 17 the glands had broken down previous to the operation and the condition of the parts became practically that of an abscess, which on being opened and scraped out healed up by union of granulation tissue without further suppuration. In case 16 the glands had not broken down. As far as possible they were removed and a large part of the wound healed by primary union, but a small portion suppurated on the surface where there was free drainage. In case 18 after the operation the central part of the wound did not unite on account of some bleeding beneath the surface; this part healed by granulation, and although at first some broken down blood clot was seen there was no actual pus. In case 19 the wound, after an operation for strangulated hernia, suppurated freely. Case 20 sustained severe burns over the body and arms and was a typical example of a granulating wound.

Of these cases, cases 18, 19 and 20 will be discussed together but the remaining two cases will be taken first.



WOUNDS HEALING PARTLY BY FIRST INTENTION AND PARTLY  
BY GRANULATION.

(Cases 16 and 17.)

*The red corpuscles and hæmoglobin.*—As in the case of healing by first intention no general course appears to be followed by the red corpuscles and hæmoglobin. In case 16 there was a distinct increase of the red corpuscles after the operation, but this no doubt is accounted for by the considerable amount of bleeding which took place during the operation, as the increase was only temporary, the red corpuscles soon regaining their normal numbers. Later on, the patient became distinctly anæmic as will be seen by the course of the hæmoglobin, and his appearance on discharge also confirmed this observation.

In case 17, which approaches very closely to a case of primary union, there was a diminution of the red corpuscles after the operation with a slight increase later, thus simulating the conditions which existed in cases 11 and 15 in the other series.

*The leucocytes.* Case 16.—Before operation, it will be noticed that a leucopenia existed; this, together with a large proportion of lymphocytes is sometimes a characteristic of the blood in tuberculous conditions, according to Ewing and Cabot.

On the day after the operation there was a slight increase of leucocytes, but on the second day there was again a leucopenia. During the whole course of the healing process the highest recorded leucocytosis was only 10,312, on the sixth day after operation, when it was found that a portion of the wound had suppurated superficially.

Case 17.—As was naturally expected in this case, when the pus was evacuated the leucocytes dropped to normal on the day after the operation.

With regard to the changes in the various types of leucocytes observed in these two cases, as has been stated previously, before the operation a lymphocytosis existed in case 16. On the day following the removal of the glands the polynuclear cells had increased from 42·4 per cent. to 63 per cent., and the lymphocytes had diminished from 54·6 per cent. to 29·2 per cent. On the second day there was a still further diminution of lymphocytes.

From the third day onwards the lymphocytes began to increase, and the polynuclears to diminish once more, and on the eighth day after the operation a condition was established similar in many respects to that existing previous to the operation, except for the fact that the polynuclear cells were still further diminished, only being 89·6 per cent., while the lymphocytes were 53 per cent. An increase of the transitional cells made up the deficit.

As in the cases of primary union, there was an increase of the mononuclear and transitional cells after the operation, the maximum occurring on the third day, being 8·6 per cent.

Eosinophiles did not appear until the third day after the operation, and then never attained the height to which they rose in the five cases of primary union, the maximum being only 2 per cent. on the sixth day.

Mast. cells were seen frequently, some being found on the day after the operation.

The increase of temperature soon after the operation in this case was due to an ague attack, and not to the condition of the wound. In case 17 the course of the polynuclear cells and lymphocytes was similar in all respects to that taken by the same cells in cases of primary union, there being also a similar reaction of the mononuclear and transitional cells on the second day, and of the eosinophiles on the third day. All these changes seem to have taken place earlier than in cases of primary union, but otherwise the condition of the blood was almost identical.

#### CHANGES IN THE BLOOD DURING THE HEALING OF GRANULATING WOUNDS.

(Cases 18, 19 and 20.)

*The red cells and hæmoglobin.*—Case 19. As in case 15, the blood examined before the operation showed an increase of red corpuscles due to concentration of the blood caused by vomiting. Consequently, on the day after the operation a diminution of the red corpuscles was noticed, but unlike case 15 there was no increase a few days later. Instead, there was diminution of both the red corpuscles and hæmoglobin throughout the illness, a

considerable amount of anæmia being present. In case 20 the conditions were very similar, the anæmia being partially covered up by the concentration of the blood which was due to diarrhœa. The value of an estimation of the red corpuscles as a means of proving the existence of concentration of the blood is well shown in this case. On admission, the red corpuscles were 7,150,000, and the leucocytes 50,000. The drop of the leucocytes on the next day to 20,000 appeared at first somewhat remarkable, but on glancing at the red corpuscle figures this was explained. The leucocytosis of 50,000 on admission would be equivalent to about 30,000 in non-concentrated blood.

As in case 19, the red corpuscles never rose again after the first drop, but gradually diminished in numbers during the granulation of the wounds, except during the period of diarrhœa.

In case 18 there was an increase of the red cells above normal during the two days following the operation, but these cells remained high throughout and no anæmia developed as in cases 19 and 20. Attention may be called in this case to the very high hæmoglobin percentage (109 per cent.) present on the day preceding the operation. The cause of this I am at a loss to explain, in fact at first I thought a mistake must have been made in the estimation, but three separate examinations gave the same result. The only occasion besides this on which I met with such a high hæmoglobin value was in case 12, where 106 per cent. was recorded on two occasions, the patient being a very plethoric individual.

*The leucocytes during the healing of granulating wounds.*—The progressive increase of leucocytes in case 18 and the maintained leucocytosis in case 19 led to the suspicion that all was not going well with the wounds. This could not be diagnosed for certain in case 19 by the leucocytosis alone until the fourth day after operation when there was a sudden increase in these cells. But the temperature had forestalled this leucocytosis by suddenly rising to 103° on the second day, when the wounds were dressed and looked satisfactory. During the fifth and sixth days the leucocytosis fell from 22,187 to 13,750 and when the wound was dressed on the latter of these two days, pus was found to be

discharging from the lower part of it. Most probably the diminution of the leucocytes on the latter of the two days mentioned, was due to the pent-up pus having found an aperture for escape.

In case 18 although the temperature was only just above normal during the few days following the operation, the continued high leucocytosis suggested an investigation of the wound and some broken down blood clot was discovered and when this was evacuated the leucocytosis began to diminish, and although both ends of the wound had healed the centre was left open and eventually granulated.

*The leucocytes during the later stages of a granulating wound.*—In all these, cases 18, 19 and 20, the irregular course of the leucocytes, sometimes reaching nearly normal and at other times rising considerably, is a characteristic shared by all. Whether this is a noticeable feature in all granulating wounds it is impossible to say, but as these facts agree with those of Cabot's observation mentioned in the earlier pages of this paper, I am inclined to think it may be so.

In neither of the Cases, 19 and 20, in which the blood was examined very frequently during a whole month, was the blood count normal at the end of that time, although on one or two occasions the leucocytes temporarily fell to normal, or nearly normal.

*Changes in numbers of the various types of cells during the healing of a granulating wound.*—In case 18, for some reason or other, previous to operation, the lymphocytes and polynuclear cells were nearly equal in numbers, both being about 46 per cent. of all leucocytes. The blood was taken just previous to operation, when food had been withheld for some hours. Whether this or some other factor was the cause I am not in a position to say, as it is beyond the province of this paper to enter into a discussion concerning the causes of lymphocytosis. In both cases, 18 and 19, there was an increase of the polynuclear cells on the day following operation, which in case 18 was continued into the second day; but in case 19 was diminished. In the latter case

it was not until the fourth day that these cells again began to increase, namely, on the same day that the leucocytes were suddenly elevated, so that both from the course of the leucocytes and of the polynuclear cells it appeared as if the wound was doing satisfactorily until the fourth day. But the temperature chart had given an indication of the reverse two days previously, so that from an examination of the blood alone it was impossible to prognose suppuration before the temperature had shown signs of its existence. In case 18 the condition of the blood showed more than the temperature chart, the increasing leucocytosis and increasing number of polynuclear cells calling forth a strong suspicion that the wound was not healing normally. In both these, as usual, the lymphocytes diminished when the polynuclears increased.

In case 20 the polynuclears maintained a high proportion during the first four days after the accident, reaching 94·6 per cent. on the second day, the lymphocytes being correspondingly reduced.

During the process of granulation the polynuclear cells in cases 19 and 20 remained considerably above normal throughout, the figures in case 19 varying from 74·8 per cent. to 87 per cent., and on the twenty-ninth day after operation, when the condition of the blood was last recorded, were still 78 per cent. In case 20 the figures varied between 68·8 per cent. and 81·8 per cent., but on the twenty-sixth day after the accident they had fallen to 63 per cent. and were still about the same on the thirtieth day when the last count was made. In case 18 the polynuclear cells began to diminish on the fifth day (when they were 75·8 per cent.) and as in cases of primary union, maintained a steady decrease to 58·8 per cent., on the thirteenth day after operation the lymphocytes being 30 per cent.

As to the large mononuclear and transitional cells in these three cases, 9·6 per cent. was recorded as the maximum number of the two cells together in cases 18 and 19, both occurring on the second day after operation. In case 20 the maximum did not occur until the twenty-sixth day. In all three cases the changes took place chiefly in the numbers of the transitional cells, the large mononuclear being scarcely altered throughout.

*The eosinophile cells in wounds healing by granulation.*—All these cases present points of interest with regard to the eosinophile cells. In case 18 in which there was no pus formation throughout, the eosinophiles were never absent, but there was no reaction above normal, as was observed in all the five cases of primary union, the highest point reached being 3·6 per cent.

In case 19, where pus was plentiful, eosinophiles only appeared in scanty numbers.

In case 20 there was a reaction of the eosinophiles to 4·6 per cent. on sixth day after the accident, similar in all respects to those cases where healing by first intention took place, although the wounds were freely discharging pus from their surfaces during the early stages of the case. Eosinophiles were only absent on the third day after the injuries.

Myelocytes, though absent in case 18 were present in cases 19 and 20, being especially prevalent in case 19, in which they rose to 2·6 per cent.

Mast. cells were present daily in case 18, and were only found associated with eosinophiles in cases 19 and 20.

#### CONCLUSIONS CONCERNING THE CONDITION OF THE BLOOD DURING THE HEALING OF WOUNDS.

##### *The red corpuscles and hæmoglobin.*—

- (1) In wounds healing by first intention the red corpuscles appear to increase temporarily during the few days subsequent to operation, this increase, which is generally slight, being probably caused by the hæmorrhage of operation. In cases where the blood has been concentrated by vomiting, etc., previous to operation, there appears to be a diminution soon after the operation, followed later by an increase. The hæmoglobin does not necessarily increase with the red cells.
- (2) In wounds healing by granulation, where purulent discharge is present in the wound, there is no temporary increase of the red cells during the few days following

an operation, and both the red cells and hæmoglobin appear to diminish gradually throughout the healing process. Where no pus is present the red corpuscles and hæmoglobin during the healing of a granulating wound follow the same course as in wounds healing by first intention.

*The leucocytes.*—

- (1) In all cases of healing by first intention there is a slight increase of the leucocytes, in some cases above normal, on the day after operation. This increase is transient, and if above normal, generally falls gradually to normal on the second or third day. This transient increase of leucocytes does not coincide with a rise of temperature.
- (2) In cases where a leucocytosis exists previous to operation, diminishes on the day following the operation if the wound is healing; granting, that is, that there is no other extraneous cause for a continued elevation of the leucocytes.
- (3) Slight suppuration on the surface of a wound does not necessarily cause an increase of the leucocytes during the healing of the wound, if free drainage is established.
- (4) A progressively increasing or maintained leucocytosis after operation indicates that a wound is not healing satisfactorily. A sudden elevation of the leucocyte curve within a few days after operation generally means the presence of pus.
- (5) The temperature curve may indicate suppuration previous to a rise in the number of leucocytes. On the other hand, when the temperature appears to be satisfactory, the leucocyte curve may raise suspicions.
- (6) During the later stages of a wound healing by granulation the leucocyte curve oscillates above normal for some weeks, occasionally dropping to normal for a day, and then rising again. Whether this continues

until the wound has quite healed or not is uncertain, but the evidence tends to show that it does not.

*Changes in the various types of cells during the healing of a wound.—*

- (1) In wounds healing by first intention the polynuclear cells may diminish on the day after an operation, notwithstanding the existence of a slight leucocytosis. In some cases the polynuclear cells appear to increase on the day after operation, even though the leucocyte and temperature curves are normal. In cases where both the leucocyte and temperature curves are oscillating above normal, the polynuclear cells may be quite normal in numbers, or even subnormal if healing by first intention is in progress.
- (2) During the later stages of healing by first intention, there is a gradual diminution of the polynuclear cells below normal, and an increase of the lymphocytes above normal, which continues after the wound has perfectly healed. In some cases this diminution of the polynuclear cells is so great that they are even exceeded in numbers by the lymphocytes. The lymphocytes may reach their maximum before, after, or on the same day as the polynuclear cells, but in cases where the polynuclear cells have diminished on the day after the operation these cells are more likely to reach their minimum before the lymphocytes reach their maximum.
- (3) The large mononuclear and transitional cells in cases of healing by first intention are generally increased between the fourth and seventh days after operation, this increase being chiefly due to the transitional cells.
- (4) The eosinophile cells generally increase considerably above normal, as a rule between the fifth and seventh days after the operation, in cases healing by



first intention or by union of granulation tissue. This reaction of the eosinophile cells does not appear to take place if slight superficial suppuration exists, except in cases of burns.

- (5) After operation in cases in which the wound breaks down, the polynuclear cells maintain a high, and the lymphocytes a low proportion throughout a greater portion of the time during which the wound is healing, even when its surface is kept quite free from purulent discharge. That eventually no doubt they behave in a similar fashion to that followed by wounds healing by first intention is a reasonable supposition, but apparently this does not take place until some weeks have elapsed.
- (6) In cases of suppuration the increase of large mononuclear and transitional cells is only very slight and they do not appear to reach as high a maximum as in cases in which healing takes place by first intention.
- (7) In granulating wounds where no purulent discharge is present, the eosinophiles may be prevalent throughout, although there is no marked increase of a nature similar to that which occurs soon after operation in cases where the wound heals by first intention. Where pus is present in the wound, except in cases of burns, the eosinophiles are very scanty. In burns, these cells are present throughout even though the surfaces of the wounds are freely discharging pus; there may also be a reaction above normal of these cells very similar to that which takes place during healing by first intention.
- (8) Myelocytes may be especially prevalent in cases of suppuration, but are not found when healing by first intention is taking place.
- (9) Mast. cells are rarely present unassociated with eosinophiles.

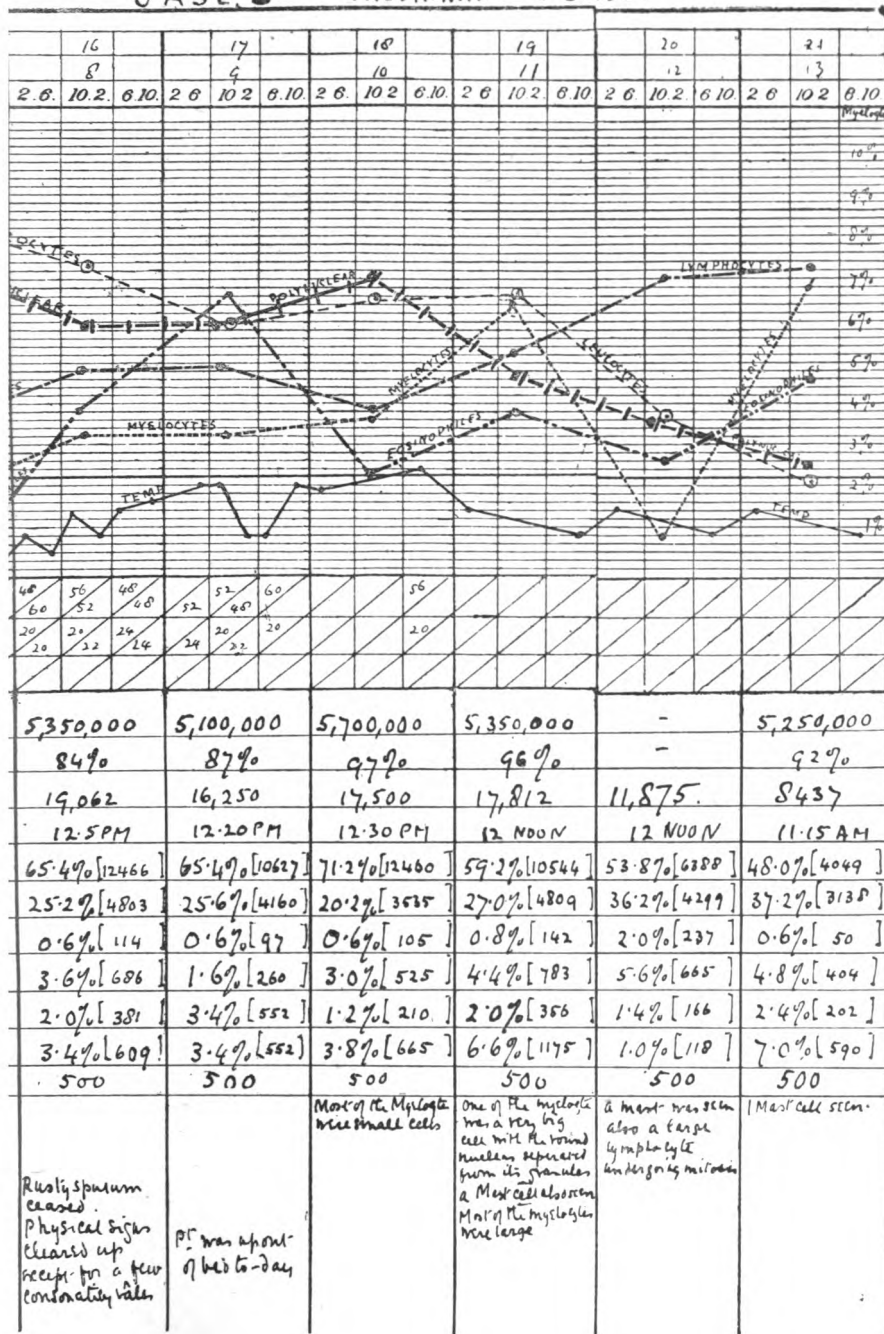
How far these observations may be true in wounds of a smaller degree is uncertain. In the cases above recorded the maximum

linear length in any case was twenty-five inches in case 11, the minimum being about three inches in case 17. In all cases (excluding case 20) the incision was carried through the skin and subcutaneous tissues. Whether slight wounds, as for instance a small cut on the finger or superficial abrasions have any general effect on the blood is doubtful, and I should think very improbable. It would be interesting to know at what point the limit is reached, also whether in cases of simple fracture the same changes would be observed as those recorded as occurring in wounds healing by first intention.

In the foregoing paper, so much space has been occupied by a record of facts elicited from the charts and tables that, without considerably adding to the present somewhat lengthy article, it would be impossible to enter into a discussion concerning the "why" and the "wherefore" of the various conditions observed. No doubt many more words might have been said and many more points in the charts discussed. Only the most obvious have had attention called to them here, and, if I have omitted anything of importance, the charts and tables are open to others to make what use they please of these investigations.

# CASE 3

# PNEUMONIA. L. BASE.



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# A CASE OF SARCOMA OF THE TONGUE, WITH AN ANALYSIS OF FORTY-THREE PREVIOUSLY RECORDED CASES.

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SINCE the year 1888 there has been only one case of this rare disease admitted into Guy's Hospital, and the object of this contribution is primarily to report this case and then, with a brief abstract of all previously recorded cases, to give a résumé of our present knowledge of the affection.

Thomas E., æt. 26 years, an army sergeant, was admitted into Guy's Hospital under Mr. Fripp, on September 5th 1899 for a swelling of the tongue. His family history was good, two brothers and five sisters all living and in good health. His grandmother is said to have died of cancer; both parents are living. The patient had, up to the onset of the present condition, enjoyed excellent health; he denied any history of syphilis. For five years he had been stationed with his regiment in India.

The history of his illness dates from April 1899 when he first found a small swelling in the upper and posterior part of the right side of the tongue, his attention being drawn to the part by some slight difficulty in deglutition. He went to the Wellington Military Hospital in India and was treated with potassium iodide and a gargle. During his stay in hospital a swelling was found in the right submaxillary region, and for this tincture of iodine and mercurial ointments were in turn applied to the skin. Both the lingual and the submaxillary swellings increased gradually but progressively in size, and after seven weeks he left the Military Hospital and was admitted into the Madras General Hospital where the treatment with iodides was pressed. The mass continued to enlarge. During July the difficulty in deglutition increased until only fluid food could be swallowed, and dyspnœa occurred and necessitated tracheotomy upon July 27th—about three months from the time the tumour was first noticed. He was then advised to come to England to see if any operative measure was advisable.

On September 5th 1899 he was first seen by us. He was then drowsy and exhausted, which he attributed to insomnia during the journey. He was a strong, well-built, healthy-looking man, but had lost weight during the last month owing to the difficulty in swallowing. He was wearing a rubber tracheotomy tube. In the posterior half of the dorsum of the tongue, mainly on the right side but encroaching on the left across the median line for one and a half centimetres, was a rounded mass which projected upwards and backwards until it reached and pressed upon the posterior part of the right side of the hard palate, and pushed the right side of the soft palate upwards and backwards, leaving but a narrow chink between the left side of the tongue and palate. This rounded elevation was the superior surface of a large tumour embedded in the substance of the tongue. The mucous membrane over it was thin, pale and shining, with numerous small vessels ramifying on its surface, but in no part was it ulcerated. To palpation the tumour was firm and elastic but no fluctuation was obtained in any part. It was not tender. The posterior limit of the mass could not be reached by the

finger, in front and laterally the boundary was ill-defined. At the right margin there was a narrow strip of healthy tongue substance. The anterior limit of the mass was three and a half centimetres from the tip of the tongue. On the inferior aspect the mucous membrane of the right side at the reflection from the floor of the mouth to the tongue was bulged forwards by the mass behind it; it appeared quite healthy and was freely movable on the surface of the tumour. The patient could not protrude his tongue beyond the lips.

A view could be obtained of the left tonsil in the narrow interval between the left side of the tongue and the palate, but the right tonsil together with the right anterior pillar of the fauces was pushed back and hidden from view.

The submaxillary region of the right side was bulged downwards by a mass which could be felt to extend from the angle of the mandible along the lower border of that bone to within two centimetres of the symphysis in front and crossing the middle line of the neck to within two centimetres of the body of the bone on the left side. This swelling was placed entirely above the hyoid bone; it was more prominent on the right side, and of smooth, rounded form, except just beneath the middle of the body of the right side of the lower jaw, where there was a rounded protrusion, firmer than the rest of the tumour. This was thought to be the submaxillary salivary gland. As upon its mucous surface so here upon its cutaneous surface the mass was firm and elastic to the touch, slightly movable from side to side, and it gave no sense of fluctuation in any part. The skin moved freely over its surface. Upon bimanual examination with the fingers of one hand on the swelling in the mouth and those of the other in the submaxillary region, the whole appeared to be one mass and could be moved slightly up and down and from side to side. No enlarged lymphatic glands could be felt in the neck.

The patient could swallow fluids fairly well and soft foods, such as bread and milk, with a slight effort. He could phonate when he blocked the opening of the tracheotomy tube, but no

laryngoscopic examination could be made owing to the position and size of the tumour.

He had no cough and the lungs were normal to percussion and auscultation. The liver could not be felt. The testes felt normal and no scars or other signs suggestive of syphilis could be found.

For three days the patient practised feeding by means of a rubber tube attached to a spout-cup and passed to the back of the tongue. He was given a mouth wash of chinisol (1:600) to be frequently used throughout the day.

*Operation.*—September 9th, 1899. Patient was anæsthetised by means of chloroform vapour *via* the tracheotomy tube. The right side of the face and neck, which had previously been shaved and enveloped in a lysol compress, were thoroughly cleaned and the head drawn over to the left. An incision was made from one inch below the lobule of the right ear downwards and forwards along the anterior border of the sterno-mastoid muscle for two inches and then forwards and upwards to the symphysis of the jaw. The skin, platysma and fascia thus mapped out were turned up in a flap and the lingual and facial arteries were ligatured close to their origin. The facial artery was again secured as it turned up over the mandible and the flattened submaxillary salivary gland was removed. The hypoglossal nerve was divided as it lay tensely stretched over the tumour, and the mylohyoid and hyoglossus muscles cut through and reflected. The digastric was drawn aside by retractors. The lower part of the growth felt in the submaxillary region was thus exposed and was freed from the surrounding structures without difficulty. An opening was next made through the lateral wall of the pharynx and, on exploring with the fingers, the base of the tongue above the epiglottis was found to be free from growth and to possess a healthy covering of mucous membrane for about an inch. The pharynx was plugged with absorbent pads, anchored by silk passing through the mouth. The upper part of the tumour was next attacked, and to facilitate this being reached through the wound, the mass was meanwhile pushed downwards by fingers in the mouth. The growth here



infiltrated the lingual substance and necessitated the incision being carried somewhat widely into healthy tissue. Finally the healthy mucous membrane of the tongue around the growth was incised from the mouth with scissors and the whole mass was delivered by the cervical wound. The bleeding was never excessive and was easily controlled by forceps. A few small lymphatic glands were removed from the submaxillary region.

The pads in the pharynx were replaced by clean ones and the lateral wall of the pharynx sewn up with four interrupted salmon-gut sutures, the knots being tied on the inner side and the ends left long and drawn out of the mouth. The muscles of the tongue and floor of the mouth were sewn up with fine silk, the hypoglossal nerve was spliced, and the flap of superficial structures was brought back into place and retained by salmon-gut sutures, a small gauze drain being left in the angle of the wound. The mucous membrane of the dorsum of the tongue, together with the underlying muscle substance, was sewn up with fine silk and painted with iodoform dissolved in ether. Iodoform gauze was stuffed into the back of the buccal cavity with the object of controlling oozing into the space left by the removal of the tumour and a gauze dressing was applied to the cervical incision, and, together with a large enough quantity of wool to act as a splint to the neck, firmly bandaged.

The administration of the anæsthetic lasted two hours and a quarter and was well borne.

*Progress.*—The patient was given nutrient enemata and suppositories alternately every four hours for the first twenty-four hours, after which he was fed upon albumen water, and later milk and beef juice by means of a tube passed to the back of the mouth. The plugs in the mouth and pharynx were removed after eighteen hours.

September 12th. A small fistula was found leading from the pharynx to the lower part of the cervical incision, but it gave little trouble and soon healed. The tracheotomy tube was dispensed with on the 14th, and the pharyngeal stitches were removed through the mouth on the 16th. The further progress was very satisfactory, and on September 24th he could swallow

soft foods with but little discomfort. Owing to the paralysis of the right lingual muscles, consequent upon the division of the hypoglossal nerve, his speech was somewhat indistinct, but this improved much before he left the hospital on October 8th.

November 23rd 1899 he was again seen. The cervical wound and the interior of the mouth appeared quite healthy. His speech had further improved and his only complaint was that he frequently bit his tongue whilst eating.

Letters were received from him in July and December 1900 during which time he had been employed as drill instructor in a school. He described himself as "quite fit."

February 16th 1901 he was again seen. The lisp in his speech had almost disappeared. There was no difficulty or pain in swallowing. The tongue was wasted and flaccid on the right side and on protrusion pointed to the right, but could be moved in any way desired. The cervical scar was healthy. In the mouth there was some thickened scar tissue on the right side of the tongue opposite the last molar tooth but the patient was positive it had not increased in size during the past twelve months. The right tonsil and fauces were normal. There was no glandular enlargement. The chest remained normal to percussion and auscultation, in fact there was no sign of recurrence anywhere.

The *tumour removed at the operation* consisted of an egg-shaped mass, rather more blunt at its lower extremity, measuring seven centimetres in the long diameter and five in the transverse. If placed in the position it originally occupied in the tongue its long diameter passed obliquely downwards and forwards. Over the upper and posterior pole the muscle substance was firmly adherent to the growth and was covered with thin, stretched-out mucous membrane which was apparently quite healthy. The tumour was firm and elastic, of uniform consistence, and heavy. On section the cut surface was white, firm, of coarse texture and apparently made up in part of an intersecting network of shining bundles looking like fibrous tissue cut in various planes. There was no softening in any part, and no cysts were discovered. In the upper part there was no

distinct line of demarcation between the growth and the lingual muscle tissue covering it. The lower part was more circumscribed but had no capsule.

Microscopical sections were prepared by hardening by the corrosive sublimate method. The growth proved to be made up of interlacing bundles of fusiform cells with large spindle-shaped nuclei occupying the greater part of the cell. Between the cells was some wavy, hyaline intercellular substance, best seen in sections stained by picro-nigrosin. There were numerous thin-walled vessels between the cells. At the edge of the growth the spindle-cells were more aggregated and were infiltrated between the muscle layers which were in part absorbed.

Mr. Targett and Mr. Bellingham Smith kindly examined the sections and both of them expressed the opinion that the growth was undoubtedly a fusiform-celled sarcoma and probably one of comparatively low malignancy.

The further history of the patient is of great interest though unfortunately it is that of recurrence of the growth. He was perfectly well for more than eighteen months, but on April 8th 1901 he noticed slight pain and difficulty in swallowing. This increased rapidly and the right side of the tongue became swollen during the night of April 10th. He came up to London and was readmitted on the following day, when the right side of the tongue was found to be swollen, tense, glossy, and very painful to palpation. The right anterior faucial pillar was red and swollen, and the breath was foul. On passing the finger over the dorsum of the tongue on the right side the margins of a small ulcer could be felt. His temperature was 100·8° F. A diagnosis of glossitis was made and it was thought that there was no recurrence of the sarcoma. He was given nitrous oxide gas and the right side of the tongue and the anterior pillar of the fauces were incised without, however, finding any pus. On April 12th the tongue was more swollen and painful, temperature 101·2° F., and during the evening there occurred suddenly some purulent discharge from the lingual incision. The swelling and pain then rapidly subsided. On laryngoscopical examination a small, rounded ulcer, with a brown, sloughy base, was found on the

right side of the base of the tongue, and with the possibility of this being specific in origin iodides were given, but without any marked effect upon the rate of diminution of the ulcer which however was smaller when on April 29th he returned to his work in the provinces perfectly well in general health and locally with the exception of this diminishing ulcer.

The patient then went on well for six months, but in November 1901 he again noticed that the right side of the tongue was swollen. He at once communicated with us and was admitted to hospital for the third time on November 6th. The right side of the tongue was larger than the left and was less movable. On the dorsal surface of the right side there was a slightly elevated area, oval in shape, commencing two and a half centimetres behind the tip of the tongue. This area was confined to the right side; it nearly reached the median line and measured three centimetres in the antero-posterior diameter. The mucous membrane over it was healthy, but rested on a mass of firm consistence embedded in the lingual substance. Immediately behind the posterior limit of this mass was a shallow depression, at the lowest portion of which was a small sinus passing downwards and outwards. Again, behind this sulcus and overhanging it was a rounded mass projecting from the dorsal surface of the tongue, about one and a half centimetres in diameter, firm but painless to the touch and covered with smooth, thin mucous membrane. The finger in the mouth could not reach the surface of the base of the tongue behind this second eminence, but on examination with the laryngoscope the mucous membrane behind it appeared to be healthy. With one hand in the submaxillary region the tumour could be felt on pressure being made from above by fingers in the mouth. The patient suffered no pain and there was only very slight difficulty in swallowing solid food. The tongue could be protruded half an inch from the edge of the teeth and articulation was but little interfered with. There was no enlargement of the submaxillary lymphatic glands, nor any evidence of metastatic deposit of growth elsewhere in the body. He was again given large and increasing doses of potassium iodide but no diminution in the size of the mass was observed.

It was decided to operate again, and on November 12th an anæsthetic was given and tracheotomy performed. The pharynx was packed with absorbent pads, anchored by silk, and a stout ligature was passed through the tip of the tongue on either side of the raphé. An incision was made in the neck through the scar of the previous operation and was carried through firm cicatricial tissue until the mucous membrane of the floor of the mouth was reached. This was incised and the tongue drawn out of the cervical wound by means of the ligatures through the tip. An incision was then made into the tongue commencing at the right margin of the organ, one and a half centimetres from the tip and passing backwards and inwards to the raphé, which was split by means of scissors as far as the posterior third. Here the incision was carried outwards and the right side of the tongue thus partially removed. On examination of this piece it was found that the cut had actually passed through the growth both behind and inferiorly as shown in the accompanying diagrams by the continuous lines, so that further sections represented in the diagrams by the dotted lines were made into the healthy tissue surrounding the mass, and by this means the whole of the growth

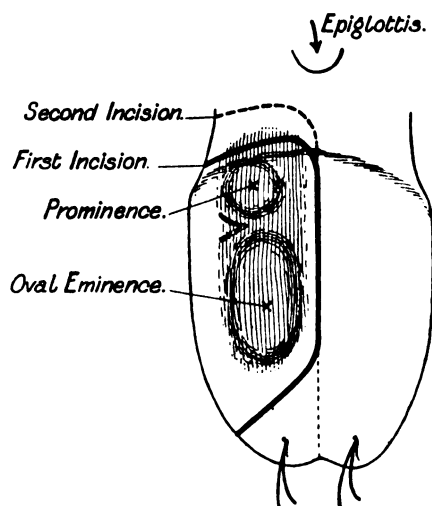


Diagram 1.

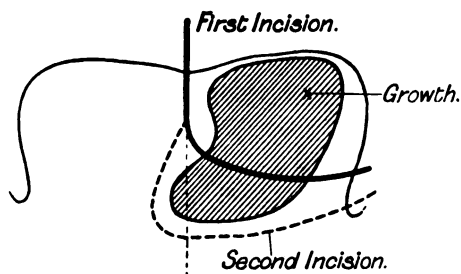


Diagram 2.

was removed together with a layer of healthy lingual tissue. The inferior part of the growth in the middle third of the tongue had crossed the middle line, pushing the muscle-planes before it. The raw surface thus left was painted with iodoform dissolved in ether and the pharyngeal pads were replaced by clean ones. The extremities of the cervical incision were brought together by salmon-gut sutures, leaving an opening for drainage at the angle of the flap, which was packed with iodoform gauze. Gauze was also stuffed into the mouth.

The patient was somewhat collapsed after the operation but improved with hypodermic injections of strychnine and saline enemata. The tracheotomy tube was removed on the third day. The cervical opening quickly granulated up. In a fortnight the patient could swallow minced foods without difficulty and when he left the hospital on December 5th 1901 he was quite well.

April 1902. A letter was received from the patient in which he said he could "feel something loose at the back of his tongue." He came up to London to see us on April 25th that is six months after his last operation. Since he left the hospital in December 1901 his health had been very good and he had put on weight. The following notes were made about his condition in April 1902:—His articulation is fairly distinct but he has difficulty in pronouncing words containing the consonants C, G, R, L, D, T and J, owing to the limitation of the movements of the tongue. From the same cause he has some difficulty in

masticating his food, but there is no interference with swallowing when once it has reached the back of the mouth. The scar of the cervical incision is puckered at the site of the old fistula, but is otherwise healthy. The tongue is small and fixed on the right side to the floor of the mouth so that protrusion can only be carried to the lower teeth. On attempt being made to protrude the organ it is seen that the muscles of the left side only are acting and there is a tendency to push the tongue to the right, but owing to its fixity the teeth on the right side cannot be reached. For the same reason he is unable to touch the upper teeth or the roof of the mouth in its anterior part. The right side of the tongue is only slightly smaller than the left, so that from its present appearance it is difficult to appreciate how much was removed at the last operation. The right faucial pillars are absent and the right upper margin of the epiglottis is in direct view; it is the epiglottis that the patient felt and thought to be loose at the back of the tongue. The mucous membrane at the floor of the mouth opposite the right molar teeth is firmly adherent to the underlying scar tissue which is directly continuous with the cervical cicatrix. There is no sign of recurrence in the mouth, no enlarged glands can be felt in the neck, and examination of the chest and abdomen reveals nothing abnormal.

The parts removed at the second operation in November 1901 were in two portions, which when placed together formed a

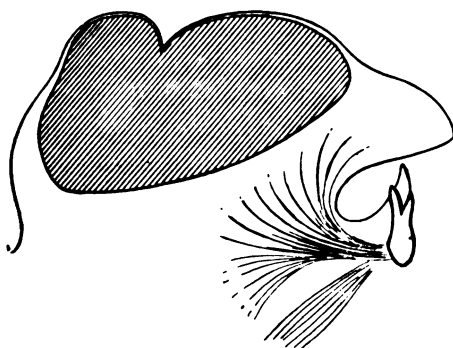


Diagram 3.

somewhat pyramidal mass with the base posteriorly and the apex reaching to within two centimetres from the tip of the tongue, occupying the position depicted in Diagram 3. The portions forming the oval and rounded prominences described above were found to be directly continuous with each other. The whole mass measured five and a half centimetres in the longest antero-posterior diameter and three and a half centimetres in the greatest vertical diameter. It was covered superiorly by thin, pale mucous membrane, quite healthy except in the position of the sinus, which was probably the result of the incision made into the tongue the previous April. The remaining surfaces of the mass were coated by lingual muscular tissue.

On section the growth had an exactly similar appearance to that removed in September 1899 (two years and two months previously), the cut surface being very firm and showing numerous shiny interlacing bundles. The tumour was not encapsuled, it could be easily separated from the muscular tissue surrounding the posterior and inferior surfaces, but elsewhere the muscle substance in proximity to the growth was very adherent and showed no definite line of demarcation. As in the primary growth there was neither softening nor cystic formation in any part.

On microscopic examination exactly the same features were presented as already described in the primary tumour, the same arrangement of fusiform cells in interlacing bundles and the same tendency to infiltrate between the planes of muscle fibres. Numerous thin-walled vessels were present amongst the cells and the intercellular substance was wavy and structureless.

Our case is therefore one of a fusiform-celled sarcoma of the tongue of comparatively low malignancy, recurring in situ after a lapse of two years, without any enlargement of the cervical lymphatic glands or any sign of metastatic deposits in any other part of the body.

No detailed description of this disease is to be found in the most modern text-books on surgery. If any mention of it is made it is cursorily dismissed in a very few words. In their



recent edition of the "Diseases of the Tongue," Butlin and Spencer devote a few pages to the affection but are silent as to the diagnosis and treatment of the disease. For the purpose of this communication all the available literature on the subject has been reviewed and we hope, by a comparison of all the recorded cases, to indicate some points which may in the future prove useful in the diagnosis of a doubtful lingual tumour.

Under the designation of "Sarcoma of the Tongue," we have found the reports of forty-three cases, the earliest of which was published in 1864, and to these must now be added the case we have just recorded. Of these forty-four cases by no means all can now be accepted as true examples of the disease, for tumours that have no real pretensions to be sarcomata have evidently been included under this title. In some the reports are so meagre, even to the omission of a microscopical examination of the growth, that but little value can be attached to them; whilst in others the history after the operation is so short as to render them valueless as a basis for any conjecture as to the ultimate prognosis. Direct communication with the respective authors has however elicited replies which have gone far to remedy the latter defects in the reports, and we are much indebted to those who have been good enough to reply to our letters. We have excluded from our table of true sarcomata of the tongue all those cases upon which there appears to us to be room for doubt as to the nature of the growth, but abstracts of these rejected cases are appended at the foot of this paper, together with our reasons for the exclusion of each. In this manner ten cases have at once been put aside as being of too doubtful a nature for classification, whilst another, reported by Godlee in the Transactions of the Pathological Society, must be placed in a separate category, as it proved on examination to be an adeno-sarcoma arising in the small mucous gland in the side of the tongue described by Blaudin and Nuhn, wherefore it cannot be included among the malignant tumours arising in the connective tissue substance of the organ. Of the remaining thirty-three cases, the claim of three to rank amongst lingual

tumours rests solely upon the direct extension to that organ of a sarcoma commencing in the tonsil, whilst a fourth is obviously a case of secondary metastatic growth, the primary lesion being a sarcoma of the lower end of the œsophagus. Twenty-nine cases of true primary malignant connective-tissue tumours of the tongue remain for tabulation, and though fully aware that this is a small number from which to generalise, the great similarity in the clinical details of these cases gives us reason to hope that the following picture of sarcoma of the tongue will be found in the future sufficiently true to justify its presence in this volume. Doubtless there are many cases of this affection that have not found their way into literature, and perhaps many more that have gone to the grave unrecognised, for it is a singular fact that the majority of cases have been recorded during the twelve years that have elapsed since the appearance of Mr. Targett's short monograph in the Guy's Reports of 1890.

*Etiology.*—Sarcoma of the tongue is undoubtedly a rare disease and it is somewhat remarkable that there should be as many as three cases recorded in the Surgical Reports of Guy's Hospital during the last thirty years, all of which are included in our list of true sarcomata.

The *age* of the patient varies within wide limits, the youngest being seven weeks and the oldest sixty-five years of age. Thus in twenty-five cases in which the age is given, there occurred:—

Under 15 years	...	...	...	...	3 cases.
Between 15 and 30 years	...	...	...	...	9 "
" 30 " 45	"	...	...	..	7 "
" 45 " 55	"	...	...	...	0 "
Over 55 years	..	...	...	...	6 "

The *sex* of the patient, in twenty-five of the cases in which it is stated, shows seventeen males to eight females, or a ratio of only slightly more than 2:1 in favour of males, which is a much smaller proportion than that found in carcinoma of the tongue.

The influence of *traumatism* in the production of the growth is suggested by Dunham's case, where the tumour appeared at the situation of a bite of the tongue; in Littlewood's, following upon a scald; and in Marion's, in which it grew at the point of

contact with a jagged, carious dental stump. The genesis of sarcoma from a benign tumour is illustrated in Perkins' case, in which a mass had been known to be present in the tongue for many years. The sudden and rapid increase in size of a hitherto benign tumour is analogous to that which occurs in other parts of the body and, as Perkin's says, "its location in the tongue, exposed to mechanical irritations from slight traumatism in the process of mastication and swallowing, may be regarded as a causative factor in the transition from a benign to a malignant neoplasm." Another case, reported by Hutchinson, is probably analogous to those in which hypertrophic structures, such as cutaneous moles and nævi, may take on a sarcomatous development, for here there was an area of rough papillary mucous membrane covering a lingual tumour and increasing rapidly in size during the year in which it was watched before operation.

Secondary metastatic deposits of sarcoma in the tongue are rare, only one case being recorded, but it is by no means so uncommon for the tongue to be directly invaded by extension of a growth commencing in the tonsil or other neighbouring structures.

*Pathological anatomy.*—The growth may occupy any part of the tongue and has been described as arising in the connective tissue substance of the base, sides, or tip, bulging and thinning out the mucous membrane covering it on either the superior or the inferior aspect of the organ. Either side of the tongue may be affected, the growth at first being limited to one side, but later extending across the middle line either by direct invasion of the tissues or, as in our case, by pushing the septum before it. In our list of twenty-nine cases, thirteen are stated to have commenced on the left side and seven on the right. With regard to the position of the growth in the tongue thirteen were found in the middle third, ten in the posterior, and four in the anterior third.

In all but two of the cases the tumour was *interstitial*, that is embedded in the muscular substance of the tongue, and, by its growth, either directly invading the surrounding tissues so that on section no definite line of demarcation could be said to exist

between the tumour and the healthy tissue, or, in those of a comparatively low malignancy, pushing aside, and thus forming for itself a pseudo-capsule of, the compressed tissues around, and remaining circumscribed and sometimes capable of easy enucleation. This apparent capsule has in some instances raised a doubt as to the tumour being in reality malignant but recurrence of the growth has taken place after a simple enucleation and it has been found microscopically that sarcoma cells were present between the planes of muscle fibres surrounding the mass. These peripheral malignant cells must be left behind after a simple enucleation. Growing from the connective tissue in the tongue the mass reaches the mucous membrane which becomes raised and stretched over it. The mucous membrane is sometimes slightly hyperæmic but seldom involved in the growth or ulcerated unless it is destroyed by pressure upon a carious tooth. The healthy condition of the mucous membrane covering a lingual sarcoma often affords an important sign in the diagnosis of these cases, but it is only in the inferior aspect of the tongue that the membrane will be actually movable on the subjacent tumour for on the superior aspect the covering is normally bound down to the underlying tissues and thus does not glide over the surface of a growth projecting in this dorsal region. In the majority of cases the growth projects and forms a smooth rounded eminence on the dorsum of the tongue, but in four cases it was the inferior surface which was bulged while the dorsum remained unaffected, whilst in five others the mucous membrane of both superior and inferior surfaces was raised. In four cases in which the growth originated in the middle and posterior portions of the tongue it had extended downwards in the muscular attachment of the organ to such a degree as to form a swelling in the submaxillary region.

A *pedunculated variety* of lingual sarcoma has been described by Marion and Lichtwitz but it is probable that in these the growth commenced in the submucous connective tissue and, extending in the direction of least resistance, carried the mucous membrane in front of it and thus formed a short thick pedicled mass. The mucous membrane on the surface of the tumour is

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very much thinned by stretching and if rupture occurs, as may happen from contact with the teeth or with food, rapid fungation of the growth is usual. The other cases which have been described as pedunculated sarcomata by Mercier, Mikulicz, and Melchior-Robert, are not included in this account owing to their doubtful nature.

The size of the tumour varies from that of a small nut to that of an orange. In the case reported above the mass measured seven centimetres in diameter, whilst in that described by Porcet it weighed four hundred grammes.

*Microscopical anatomy.*—The small and large round-celled and the fusiform-celled varieties of sarcoma have each been described as existing in the tongue, but the small round-celled growths are the most frequent. The tumours arising in the base of the tongue are chiefly of the small round-celled variety, but this type is by no means restricted to this situation. It is often a matter of considerable difficulty to differentiate histologically between a lymphosarcoma and hypertrophied lymphoid tissue, a fact of peculiar importance in the microscopical diagnosis of lingual tumours owing to the large amount of lymphoid tissue occurring at the base of the tongue. The confusion between the two has been amplified by the use of the term "lymphadenoma" by Continental pathologists to include tumours made up of small round cells supported by a reticulum of connective tissue.

Now small round-celled sarcomata present, in common with the other varieties of this growth, a small but variable amount of intercellular substance which can be picked out by special methods of section staining, as, for instance, by picro-nigrosin. Marion, in an article on this subject in the "*Revue de Chirurgie*," goes so far as to hold the view that cases of lingual tumour consisting of small round cells supported by any intercellular reticulum should be always regarded as lymphadenomata arising in the lymphoid tissue at the base of the tongue, and not as sarcomata. That lymphadenomata occur in this situation is undoubted, but we submit that those tumours which are of rapid growth, which do not diminish in size by treatment with arsenic and iodides, and which recur in situ or elsewhere

after removal, must be grouped among the sarcomata. In the twenty-nine cases appended fifteen are stated to be round-celled sarcoma, eleven spindle-celled, and one mixed round and spindle-celled. In three cases there was enough intercellular reticulum to justify the term fibro-sarcoma.

The majority of the recorded cases give but a very meagre account of the microscopical examination but in most of them mention is made of the presence of numerous capillary blood-vessels permeating the growth in close apposition to the cells, and of the sarcomatous elements at the periphery of the growth penetrating between the adjacent planes of muscle fibres, which appear degenerated and in part absorbed. The mucous membrane is in nearly all cases stated to be healthy, showing no epithelial downgrowth nor papillary change. In the two pedunculated cases the sarcoma cells could be traced from the tumour through the pedicle into a small mass of growth in the tongue immediately subjacent to the attachment of the pedicle.

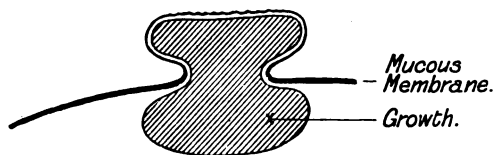


Diagram 4.

*Symptoms.*—The commencement of the growth is insidious and painless, the patient's attention being first drawn to the tongue by some inconvenience of articulation, mastication or deglutition. If the growth is situated in the *anterior part of the tongue* there is a localised increase in volume by a tumour embedded in its substance and the patient's attention is apt to be first attracted to it by the fact that he frequently bites the affected side. The tumour increases more or less rapidly in size, causing an elevation on one or both aspects of the tongue; the mucous membrane over it is stretched and often displays numerous ramifying blood-vessels. The mucous covering is not incorporated in the mass and on the inferior aspect will be freely movable on the surface

of the tumour, while on the dorsal aspect it is, as in the normal tongue, bound down to the underlying structures. The tumour is firm and elastic to the touch, gives no sense of fluctuation, and its border is ill-defined.

If the growth arises in the *middle third of the tongue* it usually projects on the dorsal aspect, presenting features in common with those mentioned above, altering the shape and volume of the organ according to its position and size. Whereas tumours in the anterior portion of the tongue are early noticed by the patient those in the middle third usually attain a greater size before attracting attention, when, upon examination, the mass may be found to be already pushing upon the soft and the posterior part of the hard palate, hiding the faucial pillars of that side and causing some difficulty in deglutition. By its extension downwards between the muscles of the tongue the growth may push forward the mucous membrane of the floor of the mouth at its reflection on to the inferior surface of the tongue, and by its further increase in size it may cause a distinct bulging in the submaxillary region. It can then be grasped bimanually by the fingers of one hand in the mouth and of the other beneath the mandible. The submaxillary bulging is unilateral and forms a smooth, rounded mass which may extend beyond the median line of the neck. The tumour is everywhere firm and elastic in consistence, and is covered dorsally by normal mucous membrane. The movements of the tongue are limited, especially that of protrusion, causing some alteration in speech, and if large the mass may cause difficulty in swallowing or in respiration.

A growth of the *posterior third of the tongue* causes usually no symptoms until it has attained sufficient size to interfere with deglutition. The patient first notices difficulty in swallowing solid food, and this disability increases until only fluids can be taken. Later there is superadded difficulty in respiration, with perhaps attacks of dyspnoea. Upon examination of the buccal cavity it may be that nothing abnormal is seen and that it is not until a finger is passed into the oro-pharynx that a tumour is found projecting from the posterior surface of the tongue into the pharyngeal cavity. As in the anterior two-thirds of the tongue

the tumour is firm and solid, covered by normal mucous membrane, and palpable from the outside below and behind the angle of the lower jaw. The voice is altered, but a laryngoscopic examination is prevented or obscured by the mass.

The *mucous membrane* covering a lingual sarcoma is in most cases thinned and healthy. In the four cases in which it was ulcerated this was probably due to contact with the teeth or with food during mastication. The ulcers presented no characteristic feature, the edges were neither raised nor thickened nor the bases excavated. They bled readily on manipulation and during mastication.

In whatever portion of the tongue the growth starts its increase in size, in spite of drug treatment, is rapid and progressive. The length of the clinical history in twenty cases varied from two months to two years, and gave an average of six months' history before operation.

The pedunculated variety of lingual sarcoma described by Marion and Lichtwitz consists of a small, rapidly-growing, flattened tumour attached to the tongue by a short, thick pedicle, through which an extension of the growth can be traced to a mass placed in the tongue under the mucous membrane at the point of attachment of the pedicle. (*Vide* diagram 4.) The mucous membrane around the pedicle is healthy, but over the surface of the projection it is thinned and, in Marion's case, was ulcerated from contact with the teeth. It is probable that in these cases the growth originates in the sub-mucous tissue and, extending in the direction of least resistance, pushes the mucous membrane in front of it and thus becomes pedunculated.

The *lymphatic glands* in the submaxillary space were enlarged in six cases out of the twenty-nine, and in three of these they showed sarcomatous infiltration on examination after removal. It is noteworthy that while of these six cases presenting glandular enlargement three were accompanied by ulceration of the mucous membrane of the tongue, yet the glands were sarcomatous in two, and purely inflammatory in only one. Of the remaining three cases presenting glandular enlargement without there being any ulceration, in only one is the nature of the enlargement



stated and then it was sarcomatous. In eleven cases only is it mentioned that there was definitely no glandular affection, but it is probable that had they been enlarged in any of the remaining cases it would have received mention in the report. After removal of the primary lingual sarcoma recurrence has been noted to occur once in the cervical glands alone, and on four occasions in the glands as well as in the site of the previous operation in the mouth. It may therefore be said that secondary deposits in the lymphatic glands are exceptional but may occur with sarcoma of the tongue.

The *submaxillary salivary gland* was found enlarged in three cases, in two of these it was removed and examined microscopically and the enlargement was proved to be merely inflammatory, while in the third case it was left in situ at the operation for the lingual tumour and it regained its normal size in a fortnight. It is possible that in these cases the enlargement is due to pressure on Wharton's duct by the growth in the tongue.

*Pain* in the tongue is exceptional, and if present is usually accompanied by, and due to, ulceration of the lingual mucous membrane.

*Hæmorrhage* may occur but only in those exceptional cases where there is ulceration.

*The general health* of the patient remains good until secondary visceral deposits have appeared, or until the mass has attained sufficient volume to interfere with deglutition or respiration.

*Diagnosis.*—The functional troubles due to the volume of the tumour, viz. the limitation of movements of the tongue and the consequent difficulty in speech and deglutition cannot be said to be especially marked in sarcoma, and the diagnosis of this disease must be based chiefly upon a knowledge of the clinical history of the case and upon the elimination of tumours that might simulate it. Histological examination of a small piece removed from the growth would prove of great value in doubtful cases, but such a proceeding is likely to be followed by a rapid fungation if the growth is malignant.

Perhaps the most common lingual tumour with which sarcoma is likely to be confounded is *gumma*, especially when the latter is single

and deeply embedded in the tongue. A gumma of this character placed in the middle or side of the tongue may cause a rounded elevation on the dorsal aspect covered by thin, smooth, and healthy mucous membrane. Such a gumma may occur at any age, even in a congenital syphilitic child, and present a firm and somewhat ill-defined mass much resembling a sarcoma. But in a short time a gumma shows signs of degeneration, the mucous membrane over it becomes pale and yellowish and eventually it gives way leaving an ulcer with ragged undermined edges and a deep sloughing base. If seen before ulceration has taken place the association with other syphilitic lesions together with a history of the disease and the rapid diminution in size of the tumour under large doses of potassium iodide will point to its gummatus nature. Lastly gummata of the tongue are frequently multiple, especially the superficial variety, whilst sarcoma has in all the cases hitherto reported been single.

It could only be quite an early stage of *carcinoma* of the tongue that might be confused with sarcoma. If the tumour is placed on the inferior aspect of the tongue the free mobility of the mucous membrane at once excludes carcinoma; if on the dorsum a carcinoma shows more definite fixity of the mucous membrane, with infiltration extending into the lingual substance, whereas a sarcoma is embedded in the tongue proper and can be felt to be independent of the mucous covering, which however, as already explained (p. 104), is not in this situation freely movable over the growth. In cases in which the tumour has increased to a more considerable size there is little chance of error, for in a carcinoma of such standing the mucous membrane is almost certain to have given way leaving a characteristic epitheliomatous ulcer with hard, uneven and everted edges. Where the mucous covering of a sarcoma has given way the resulting ulcer has ragged edges and is placed upon the surface of a tumour embedded in the actual muscular substance of the tongue. If there is any reasonable room for doubt a microscopical examination of a small piece can be made, provided only that the patient is willing to submit, if found necessary, and at once, to a radical operation. Two other facts to which some weight may be attached are the

comparative rarity of sarcoma and the different ages at which each disease occurs, sarcoma being most common between fifteen and thirty years, carcinoma between forty and sixty years. Another factor, though not so important as the age, is the sex of the patient, for whereas carcinoma occurs seven times more frequently in males than in females, sarcoma is only twice as frequent.

Other tumours of the tongue to be considered in the diagnosis are in themselves almost as rare as sarcoma, but mention must be made of cysts and benign growths. Of the *cysts*, a dermoid is nearly always situated in the median line, projecting both into the floor of the mouth and in the suprahoid region, forming a clearly defined, globular, tense and, indeed, often non-fluctuating swelling. Mucous and parasitic cysts are recorded, but, unless translucent or fluctuating, could only be diagnosed by an exploratory puncture or incision. A chronic abscess lies just under the mucous membrane, is small, moderately soft and fluctuating and usually placed on the dorsum of the tongue in front of the circumvallate papillæ. It is of very slow development and may be associated with tuberculous lesions elsewhere in the body. *Actinomycosis* of the tongue has been recorded by Hebb, forming a tumour in the tongue. It is very rare, of slow development and finally softens and ulcerates. On incision, the contents would show the yellow granules of the fungus.

*Innocent solid tumours*, such as fibromata and lipomata, are extremely slow in their growth, sometimes taking ten or fifteen years to reach the size of a small walnut and are hence readily distinguished from sarcoma. The colour of a sarcoma more nearly resembles that of the surrounding tissue than is the case with benign growths, and whereas a sarcoma in its growth will alter the contour of the surrounding parts, a benign tumour is more apt to conform to its surroundings.

*Prognosis.*—It is difficult to formulate any reliable rules for guiding the ultimate prognosis from the comparatively small number of cases that have been reported. Of the twenty-nine cases twenty-five were operated upon, with the result that eleven recovered and were known to have been well for a

varying period. We have attempted to obtain the post-operative histories of all these cases and we find that they have been free from recurrence for the following periods—13, 12, 9, 4, 4,  $3\frac{1}{2}$ , 3, 2, 2,  $1\frac{1}{2}$  years and 6 months. The first seven at least may be pronounced cures. In thirteen cases, that is in more than fifty per cent. of those operated upon, recurrence took place, necessitating further operative measures. Of these, three were well for periods exceeding two years, nine eventually died from recurrence, whilst the remaining case is the one reported above, and this has remained well after an operation for recurrence eleven months ago. Of the nine cases that died from recurrence of the growth, three died in a short time from rapid spread of the disease and the others after an interval of 9 months,  $1\frac{1}{2}$ ,  $2\frac{1}{2}$ ,  $2\frac{1}{2}$ , 3 and  $3\frac{1}{2}$  years respectively from the first onset of the disease. One case was deemed inoperable when first seen and died soon after, another died from pneumonia eight days after operation and in the remaining four the after-history cannot be obtained. Of the thirteen cases in which recurrence took place, it was in nine instances at the site of the previous operation, in one case in the lymphatic glands of the neck alone, and in four in both the original site and the glands. The immediate cause of death was found to be secondary metastatic deposits in the brain in one case, in the lungs in two, and in the peritoneum in two cases.

Though the behaviour of sarcomata elsewhere in the body might lead us to expect that the cases of the fusiform-celled variety would show a better prognosis than those of the round-celled type, yet the differences in the results in our twenty-nine cases are by no means striking in this respect. Of twelve cases of the spindle-celled variety four recovered and remained free from recurrence for periods of 12, 4, 2 and 2 years respectively, six recurred within two years, two of them dying shortly after the operation, and one in  $1\frac{1}{2}$  years from cerebral metastasis. In the round-celled type, four cases were free from recurrence for longer than three years, six recurred within two years of the operation, and five died at varying periods ranging from nine months to two and a half years. Only one death was imme-

diately attributable to the operation, and that was caused by pneumonia after a week's interval. One case was deemed inoperable from the first and quickly died.

*Treatment.*—One point which appears to be very emphatically demonstrated by a perusal of the cases we have collected is that these tumours should be *widely* removed by an incision into the healthy lingual tissue well clear of the growth, for though the ease with which the obvious new growth can sometimes be enucleated is very tempting, yet such a method of separation from the surrounding compressed structures is extremely liable to be ineffectual in removing all traces of the growth, and the pseudo-capsule remaining will contain the nucleus for a recurrence at a later date. In the case of a quickly growing sarcoma which has invaded the tongue extensively no doubt the more drastic measure of excision of the anterior two-thirds or of the whole organ would give the patient a better chance, despite the increased risk of the operation itself. The question which method of operation is most applicable depends on the nature of each individual case, and among other things upon the situation and the volume of the tumour. Small tumours of the anterior part can be attacked from the mouth, some placed in the middle third of the tongue can be more easily reached by dividing the cheek, but in those cases in which the growth has extended downwards in the muscular attachment of the tongue and can be felt in the submaxillary region, whether projecting in front of or behind the circumvallate papillæ, a supra-hyoid incision is necessary, opening up the floor of the mouth or the pharynx as the case demands. Such operative details as preliminary ligature of the lingual artery or tracheotomy will be governed by the same rules as apply in operations for lingual epithelioma.

If recurrence should ensue the secondary tumour must again be removed and widely.

Any enlarged lymphatic glands present when the case is first seen should be removed at the operation or at a later date, for although the enlargement may be purely inflammatory, yet they may be affected with sarcoma and it is impossible to determine this from their clinical aspect. Sarcomatous enlargement of the

glands of the neck following the removal of the primary lingual growth makes the prognosis grave, but they should be removed if possible for some of our cases (notably 17 and 18) show a most satisfactory ultimate result even after two operations for removal of such secondary growths.

For those cases which are deemed inoperable either from the first or owing to the extensive degree of recurrence treatment must be directed to the alleviation of symptoms—such as dysphagia, asphyxia, and pain. The only remaining hope lies in a recourse to injections of Coley's fluid, but (with the exception of Case 40 which we have not felt justified in including) there is no instance of a lingual sarcoma amongst the published cases in which treatment by any form of injection has been tried.

*Classification.*—It will be seen that the cases of sarcomata arising in the muscular substance of the tongue fall naturally into the following groups, which we have somewhat modified from the classification used by Butlin and Spencer in their new edition of "Diseases of the Tongue."

(a) *Sarcomata of a relatively benign character.*—Cases 1 to 11 in our list seem to have been of this nature. They cause an elevation upon one or both surfaces of the tongue without glandular enlargement. They are comparatively slow in their growth, are circumscribed but show no distinct capsule, and are usually separated with ease from the surrounding healthy tissue. As has already been pointed out they may recur in situ after simple enucleation from their bed, so that it is most advisable to cut wide into the healthy tissue around when operating for their removal. Microscopically either the round or spindle cells may predominate.

(b) *Sarcomata of a more malignant form.*—Cases 12 to 20.—These are almost certain to recur after removal and to require further operation at an early date, unless the primary operation has been extensive and carried wide of the growth. They are prone to involve the submaxillary or cervical lymphatic glands. We have shown on p. 108 that the lymphatic gland enlargement may be due to sarcomatous infiltration even when there is no ulceration of the mucous membrane over the primary tumour,

and that recurrence of the growth in the glands may be unaccompanied by any fresh growth in the mouth after removal of the primary mass. The cases in this class always show a much more rapid enlargement of the primary tumour, and usually an early recurrence, either in situ or in the glands of the neck. In the majority of cases in which the after-history has been obtained they are seen to have quickly ended fatally. The small round-celled variety of sarcoma is the common form in this class.

(c) *Sarcomata originating at the base of the tongue*, that is to say in that part of the organ, behind the circumvallate papillæ, which is developed by two lateral halves from the second and third visceral arches, as distinct from the anterior two-thirds of the tongue in front of the foramen cæcum, which is derived from the median tuberculum impar. Cases 21 to 27.—These growths are distinguished by position rather than by other features from the classes mentioned above, and they may be either of the round or spindle-celled form. They have in most instances already attained a large size before anything wrong is perceived by the patient, and therefore they have required an extensive operation for their removal. They show varying degrees of malignancy, and the particular tendency they manifest to recur locally is possibly attributable to their extensive infiltration into the neighbouring structures before any operation is undertaken.

A separate class has been made of these cases because confusion has arisen between them and the lymphadenomatous tumours originating in the lymphoid tissue of this part. This latter growth may form a mass steadily growing in the base of the tongue and accompanied by glandular enlargement in the neck, but arrested in development and disappearing under treatment with drugs, such as arsenic or iodide of potassium, whilst a sarcoma will be but temporarily if at all arrested by such means. Microscopical examination may afford very little distinction between such an innocent lymphatic enlargement and a small round-celled sarcoma, so that the diagnosis in these cases must rest mainly upon clinical evidence (*vide* p. 105).

(d) *Sarcomata following upon lymphangiectasis*.—The cases described by Eve and Perkins (28 and 29) appear to have been

preceded by lymphangiectasis from which the malignant growth started.

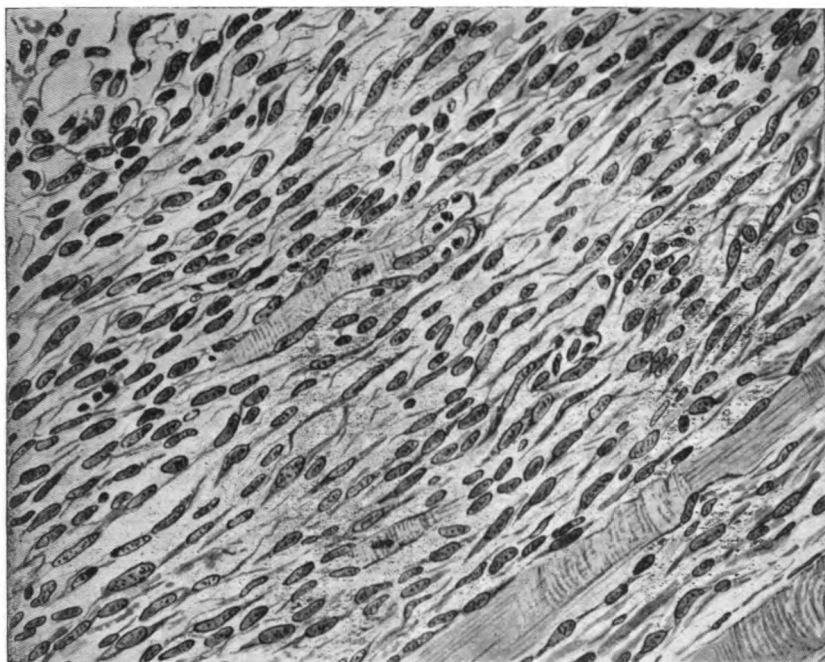
(e) *Sarcomata arising in the glandular elements of the tongue* as in Godlee's case (30) in which the growth commenced in the mucous gland of Blaudin.

(f) *Secondary sarcomata in the tongue*.—Only one case of true secondary sarcoma of the tongue can be found and in this the primary growth was situated in the lower end of the œsophagus. In another case reported as a secondary growth it is not at all clear that the tumour in the tongue was not a direct extension from the primary growth in the tonsil.

In conclusion, we wish to thank Mr. Targett and Mr. Bellingham Smith for very kindly examining the sections of the growth in our case, and Dr. Stevens for his excellent drawing of the same.



*A Case of Sarcoma of the Tongue, with an analysis of  
forty-three previously recorded cases.*



THOS. G. STEVENS, del.

A section through the periphery of the growth, showing the infiltration of the growth between the muscle-fibres.



## ABSTRACTS OF THE CASES OF SARCOMA OF THE TONGUE PREVIOUSLY REPORTED.

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Heath. *Trans. Path. Soc.*, 1869, vol. xx., p. 157.

CASE 1.—Male, *æt.* 60 years. History of six months' duration. Between the tongue and the floor of the mouth on the left side was a ragged, ulcerated surface, under which could be felt a large mass in the substance of the tongue, reaching beyond the middle line and to within two inches from the lips. There was constant pain in the tongue, but otherwise the health was good. No glandular enlargement in the neck or submaxillary region. September, 1868, the anterior half of the tongue was freely removed. There was no recurrence and the patient was in good health twelve years later. The growth was submitted to the Morbid Growths Committee, who reported it to be small, distinctly circumscribed, of an opaque white colour and prolonged downwards as a homogeneous and solid mass into the muscular attachment of the tongue. The cells were oval and in some cases elongated. There is no mention in the report of epithelial ingrowths or bird-nest formations or of any alveolar arrangement of the cellular elements. An accompanying diagram represents a small spindle-celled sarcoma.

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Butlin. *Lancet*, 1887, vol. i., p. 623.

CASE 2.—C. J., male, *æt.* 40. Swelling on left side of tongue. Two months previously there was some soreness in the left side of the tongue, but the tumour had only been noticed for one month, during which increase in size had been rapid. There was pain only on mastication. No history of syphilis. He was a strong, apparently healthy man. In the substance of the left half of the tongue was a smooth, soft, elastic swelling, bulging on both the superior and inferior aspects of the organ. It extended from the junction of the anterior and middle thirds to the posterior part of the tongue, was distinctly limited to the left side, and of about the size of a fives ball. There was no ulceration, the mucous membrane being healthy. Protrusion of the tongue was limited. The left submaxillary gland was enlarged. Large doses of potassium iodide were given, but no diminution was noted in the size of the tumour, which on puncture yielded only blood. Under an anæsthetic the tumour was incised and proved to be solid, and accordingly the left half of the tongue was removed well behind the growth. In twelve days the enlarged submaxillary gland had regained its normal size. The growth consisted of a rounded, soft, white tumour, embedded in the muscle substance of the tongue; it was circumscribed and had the appearance of a capsule at one part; it measured 3 by 2½ c.m. It was composed of small round sarcoma cells with a homogeneous intercellular substance. There were numerous small blood vessels. The patient was seen up to four years after the operation and was in good health with no sign of recurrence of the growth.

Dunham. *American Journ. of Med. Sci.*, 1895, p. 259.

CASE 3.—Male, æt. 61. Heavy smoker. No history of syphilis. A brother and sister had died of tuberculosis. Eight months previously the patient bit his tongue, causing a blister to appear, which did not heal for some time owing to irritation by a decayed tooth. Three months after the injury a hard mass was noticed under the sore area and it gradually increased in size. The tumour was embedded in the tongue substance, raising the mucous membrane of the right side for an area with a diameter of three-quarters of an inch, commencing one inch behind the tip. It was globular in shape, firm to the touch, and the mucous membrane over it was not ulcerated but smooth, save for a few prominent papillæ. There was no enlargement of the cervical lymphatic glands. After removal the growth was found to be spherical, of a pinkish colour, moderately firm and apparently uniform in structure. The mucous membrane covering it was thin, but entire. Microscopically the mucous membrane rested on connective tissue, and in no place was there any down-growth from the surface epithelium. The cells were large and round, lying in a delicate, scanty reticulum; the nuclei showed active division. Numerous small blood vessels were present. At the periphery of the growth, the sarcoma cells penetrated between the planes of muscle fibres. We have been unable to obtain any account of the actual operation performed or of the post-operative history of this patient.

Murray. *Annals of Surgery*, 1895, vol. xxii., p. 271.

CASE 4.—Female, æt. 56. In December 1893 a small hard mass was removed from the mouth at the reflection of the mucous membrane from the floor of the cavity to the inferior surface of the tongue. This had been rapidly growing for a short time. In April 1894 it had recurred, and there was a firm indurated tumour involving the greater part of the right side of the tongue and a small part of the left. There was pain in the tongue, radiating to the right ear, especially when pressure was made on the mass. The tongue could not be protruded and speech was indistinct. The right submaxillary gland was enlarged. After a preliminary tracheotomy, the whole tongue, together with the right submaxillary gland was removed by the submaxillary route. The patient did well and left the hospital in five weeks. Microscopically the growth was a spindle-celled sarcoma with some clusters of round cells in parts. A year after the operation the patient was in good health and there was no sign of recurrence. The further history of this patient has been furnished by Dr. Murray. Early in 1896 there was a slight recurrence in the scar under the angle of the jaw, and a mass about the size of a gooseberry could be felt extending inwards to the wall of the pharynx; the growth was removed with a portion of the pharyngeal wall. For a year the patient did well, but early in 1897 there was further recurrence in the scar of the previous operation, and this was again removed with a portion of the lower pharyngeal wall. The patient enjoyed good health until November 1897 when she complained of headache in the right parietal region. This increased in intensity, and later there was slight right sided facial paralysis and motor weakness of the left arm and leg. Convulsive seizures followed, and after an especially severe attack, the patient remained unconscious and died in thirty-six hours in December 1897 that is to say, nearly four years from the onset of the disease. No autopsy was obtained, but from the clinical history death seems to have been due to an intracranial recurrence.

Poncet. *Revue de Chirurg.*, 1897.

CASE 5.—D. J., male, æt. 32. In December 1886 the patient presented a mass in the under surface of the tongue, pushing forward the mucous membrane at its reflection from the tongue to the floor of the mouth. It was slowly increasing in size, but the patient refused any operation. In two years it was large enough to prevent the mouth being closed, to render speech impossible and to impede both deglutition and respiration. The soft palate was completely hidden and the finger could not reach the posterior limit of the growth. The anterior 3 cm. of the tongue was free and could be moved from side to side. The mucous membrane over the tumour was healthy, adherent on the superior aspect, but on the inferior surface it glided over the subjacent mass. The submaxillary region of both sides was bulged downwards by the growth. An incision was made from the symphysis menti to the left angle of the jaw and the facial artery divided. The submaxillary gland was removed and the lower part of the tumour easily separated. The jaw was divided in the middle line and the tumour with the mucous membrane overlying it was removed via the cervical wound by incising the healthy tissue around it. The patient did well. The tumour proved to be nearly spherical, the size of an orange, and weighed 400 grammes. It was solid, pinkish in colour and of uniform consistence. The cells were small, fusiform in shape with elongated nuclei, and formed interlacing bundles. There was a firm, homogeneous intercellular substance, but no well-formed fibres. Numerous thin-walled vessels were present in the growth. In 1892, four years after the operation, the patient was quite well and presented no sign of recurrence.

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Lichtwitz. *Archiv. Internat. de Laryngol.*, 1898.

CASE 6.—M. S., female, æt. 25. History of six weeks' duration of a small tumour on the dorsum of the tongue to the left of the middle line and 2 cm. behind the tip. The tumour was of the size of a small nut and had a short thick pedicle; it was dull grey in colour, covered by healthy mucous membrane, and firm in consistence. The tumour was removed by incising the healthy tissue of the tongue freely around the base of the pedicle. It measured 9 mm. in diameter, and on section proved to be a fusiform-celled sarcoma. The mucous membrane covering it was healthy; there were numerous small blood vessels present and some small hæmorrhages into the growth. Two years later the patient was quite well and free from recurrence.

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Abbe. *Annals of Surgery*, 1894, vol. xx., p. 72.

CASE 7.—Male, æt. 26. The patient was shown before the New York Surgical Society in March 1894 by Dr. Abbe, who had operated upon him nine years previously. At the time of the operation a tumour as large as the terminal joint of one's thumb was embedded in the dorsum of the tongue and covered by normal mucous membrane. It was solid and had been gradually increasing in size for some time. It was removed by carrying an incision into the healthy tissue around it, and proved under microscopical examination to be a "pure sarcoma." There was no sign of recurrence up to nine years after the operation,

Barling. Brit. Med. Journal, 1897, vol. i., p. 297.

CASE 8.—Female, æt. 35. History of four months' duration of a small swelling on the tongue which gradually increased in size in spite of treatment with potassium iodide. There was a round, elastic swelling embedded in the middle third of the left side of the tongue, extending to the septum internally and projecting on both the superior and inferior surfaces. It was painless to the touch; the mucous membrane was everywhere normal. The tongue could be protruded easily. A small piece of the mass was removed for microscopic examination and found to be sarcomatous. The growth rapidly fungated through the incision. Complete excision of the tongue was then performed. The growth was the size of a horse-chestnut embedded in the left half of the tongue. It was distinctly circumscribed and could be enucleated from the surrounding tissues with ease; it was moderately firm in consistence. Microscopically it proved to be a round-celled sarcoma. Three and a half years later the patient was reported to be alive and well.

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Gross. Philadelp. Med. Times, April 1872, p. 272.

CASE 9.—Female child, æt. 7 months. A tumour of the tongue had been noticed for five weeks, impeding sucking and deglutition. It occupied the posterior portion of the tongue, extending forwards into the middle third. The growth was firm and dense in consistence and situated immediately beneath the mucous membrane, which retained its normal appearance except for a few fine venules ramifying over the most prominent portion. The increase in size had been rapid, and during the last few days there had been some difficulty in respiration. There was no pain. The growth was removed and found to be ovoidal in shape and of the size of an almond. Microscopically it was composed of large oval and fusiform-shaped cells, the latter arranged in a distinctly fasciculated manner. Numerous thin-walled vessels were present. No further history can be obtained of this case.

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Fripp.

CASE 10.—Reported at the beginning of this communication.

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Targett. Guy's Hospital Reports, vol. xlvii., 1890.

CASE 11.—J. P., male, æt. 65. Admitted under Mr. Howse, in 1888, for a tumour of the tongue which, when first noticed a year previously, was of the size of a horse bean, but had gradually increased. In the substance of the tongue was a firm, globular, painless, non-fluctuating swelling, most apparent to the left of the frænum and extending back to the last molar tooth. The mass could be felt bimanually in the submaxillary region. The mucous membrane over the growth was normal. There was slight limitation of the movements of the tongue, but deglutition was not affected. Antisypilitic treatment was tried for three weeks but without effect on the size of the tumour. The left half of the tongue was then removed after incising the cheek. The growth was spherical, one and a half inches in diameter, soft and vascular. It was embedded in the substance of the tongue with a well-defined margin but without a distinct capsule. It was composed of small round cells with a fine intercellular stroma. There were numerous small vessels and strands of muscle fibre between the peripheral portions of the new growth.

Fifteen months later there was a recurrence of the growth in the left submaxillary region and also beneath the right zygoma, but there was no recurrence in the mouth. No operation was performed and the patient died in July 1891, from pulmonary trouble. No post-mortem was made. The recurrence in the neck had grown to a large size.

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Targett. *Guy's Hospital Reports*, vol. xlvii., 1890.

CASE 12.—Albert W., *æt.* 2, was admitted under Mr. Durham in 1867 for a swelling on the tongue which had been noticed seven weeks. It had increased rapidly in size but was painless. There was a tumour on the dorsum of the tongue to the left of the middle line one and a half inches behind the tip. It was whitish, of soft consistency, and measured one inch in diameter. There was no glandular enlargement, no pain, and deglutition was normal. Two months later the tumour had so increased in size as to hinder deglutition, and a portion of the tongue and tumour was removed by the *écraseur*, but it was found that the growth had not been completely removed. Recurrence was very rapid, and in a fortnight the growth was larger than before, being very soft and nodulated. The patient was removed from hospital and died shortly afterwards. No post-mortem examination was allowed. Microscopically the growth was found to be a mixed round and spindle-celled sarcoma.

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Marion. *Revue de Chirurg.*, Paris, 1897, p. 668.

CASE 13.—Male, *æt.* 17. The patient had noticed that for six months he constantly bit the left side of his tongue, but had only found a tumour for the last two months. This had slowly increased in size, and in March 1896 there was on the left margin of the tongue, 3 cm. behind the tip, a mass the size of a nut. It was somewhat pedunculated and expanded into a flattened surface, furrowed by contact with the teeth. The tumour was firm to the touch, and an extension could be traced through the short pedicle into the lingual substance. The mucous membrane was normal on the pedicle but was ulcerated on the surface of the mass. There was no pain, but slight bleeding occurred during mastication. A gland in each of the submaxillary regions was enlarged. The tumour and its base in the tongue substance were removed, but the glands were thought to be inflammatory and were left in position. The deeper part of the mass was found to contain fusiform sarcoma cells infiltrating at the periphery between the planes of muscle fibres. In the pedicle the cells were more closely packed together, but towards the surface of the growth there was an admixture of polynuclear round cells from inflammatory infiltration. There were numerous thin-walled capillary vessels in the growth. The mucous membrane covering the pedicle and the deeper part of the growth was normal, but on the surface of the mass it was deficient. A month later there was recurrence in the scar with further enlargement of the submaxillary glands. A free removal was undertaken after splitting the jaw to the left of the symphysis. The submaxillary and submental lymphatic glands were removed, but were afterwards found to show only inflammatory changes, whilst the recurrent tumour in the tongue showed the same characters as before, viz., those of a spindle-celled sarcoma. Five weeks later there was again recurrence in the tongue and the mass was again removed with 2 cm. of healthy tissue around. There was no glandular

enlargement. A month after this third operation a small tumour was again detected in the scar. It increased for a few weeks, but later diminished in size and finally disappeared in six months. There is a slight element of doubt thrown upon this case by its termination. The masses removed were clearly proved by microscopic examination to consist of spindle-celled sarcoma, but the last formation was probably inflammatory.

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Littlewood. Trans. Path. Soc., vol. xlix., p. 60.

CASE 14.—J. M., male, æt. 17. In March 1896 the patient scalded his tongue, and in April there was an ulcer on the dorsum the size of a sixpence. He was given potassium iodide and mercury for three months without benefit, and in July 1896 a tumour was found occupying the middle of the tongue, hindering speech and deglutition. The mass was round and projected from the superior surface of the tongue. The mucous membrane over it was ulcerated into a patch the size of a shilling which readily bled on being touched. The tumour was firm and non-fluctuating. The submaxillary and carotid lymphatic glands of the left side were enlarged. August 1st, laryngotomy was performed and the tongue removed by Syme's method, and three weeks later the glands were excised. The growth involved both sides of the tongue, it was firm and white on section, but hæmorrhages had occurred in places. Microscopic examination showed it to be a round-celled sarcoma with many cells in active division, and very vascular. Similar appearances were present in the glands. The left tonsil and fauces soon became attacked by a recurrence of the growth and later the glands in the neck; a mass also formed in the left temporal muscle. Death occurred on December 29th 1896 *i.e.*, five months after the operation and nine months after the scald from which the history dated. No post-mortem examination was made.

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Santesson. Virchow. Hirsch. Tahresbericht, 1887, vol. i., p. 280.

CASE 15.—History of three months' growth of tumour embedded in the left side of the tongue in the middle third. It was covered by thin, stretched out mucous membrane which was adherent to the growth. A gland below the angle of the lower jaw on the left side was enlarged. The growth proved on section to be a small round-celled sarcoma. Hyaline degeneration had occurred in a small area of the growth. (We have failed to obtain the original paper reporting this case and are consequently unable to give any post-operative history of the patient.)

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Beregszaszy. *Krankheiten de Lunge*, von Butlin; Deutsch bearbeitet von Beregszaszy, 1887, p. 226.

CASE 16.—Male, æt. 42, who died in hospital of what was supposed to be an inoperable cancer of the tongue. For two months previous to his death there was pain and a swelling in the left side of the tongue so that the patient could only swallow liquid food. His speech was indistinct and the tongue could not be protruded beyond the lips. The base of the tongue and the left pillars of the fauces were hard and infiltrated by a tumour which was painful on pressure. (There is no mention in the report of any ulceration on the surface of the mass.) On post-mortem examination there was a tumour in the posterior part of the tongue, firm to the touch and hollowed out below in



the shape of a funnel. The left side of the floor of the mouth was firm and infiltrated by the growth. There was no enlargement of the lymphatic glands. Secondary deposits of growth were found in the abdominal cavity, both as isolated masses in the peritoneum and at the mesenteric attachment of the small intestine, which was in these situations half encircled by growth. Microscopical sections of the tumour showed it to be a small round-celled sarcoma.

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Mandillon. Soc. de Med. et de Chir. de Bordeaux, November 16, 1888.

CASE 17.—Male, æt. 21. No history of tubercle or syphilis. He was first seen in January 1888 for a small tumour the size of a large pea, white in colour and situated on the inferior aspect of the tongue at the side of the frænum. It increased gradually in size, and after a short time was enucleated with the thermo-cautery. In three months it had recurred and was then twice as large as on the former occasion and was removed by the knife. It again recurred and the patient was shown before the society for suggestions as to diagnosis and treatment. It was thought by those present to be a sarcoma from its rapid progress and recurrence in situ, and a wide removal was advised. This was done in July 1888 and the tumour under the microscope proved to be a small round-celled sarcoma. The patient was in good health and free from recurrence in November 1888. Dr. Mandillon writes in answer to our inquiries, that he had the opportunity of again examining his patient in 1901, finding him well and free from recurrence after a lapse of three years.

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Mikulicz et Michelson. Atlas der Kraukheiten der Mund und Rachenhöhle. II. Helfte. Tafel xxxvii.

CASE 18.—Male, æt. 57. History of six months' gradual extension of tumour on the dorsum of the tongue. There was no pain, but the speech was indistinct. The patient was thin and feeble. The anterior third of both sides of the tongue was involved by a tumour occupying the whole thickness of the organ. It was larger on the right side than on the left, the two sides being separated by a shallow depression corresponding to the median septum. Posteriorly it was marked off from the healthy tissue by a fairly definite margin. The mass was firm in consistence and covered by healthy, though slightly hyperæmic, mucous membrane. There was no pain on palpation. The mobility of the tongue was diminished by the volume of the tumour but deglutition was not affected. No glandular enlargement was made out. The anterior half of the tongue was amputated in Nov. 1884 and the patient was well in a month. The tumour proved to be a fusiform-celled sarcoma. In July 1885 recurrence had taken place in the scar in the mouth and in the cervical glands. These were removed. Six months later the glands were again found to be enlarged and were removed. In January 1888 that is to say two years after the last operation, a small ulcer was found in the scar of the tongue but this rapidly healed on the extraction of some carious teeth. His further history is unknown.

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Stern. Deutsch Med. Wochenschrift, June 2nd 1892.

CASE 19.—C. H., female, æt. 4 years, was seen in 1890 for a tumour on the right side of the dorsum of the tongue at the level of the first molar tooth. It had grown rapidly, was embedded in the lingual substance, firm to the

touch and covered by healthy mucous membrane. The tumour was excised, but in September 1891 the child was again seen for a recurrent mass in the scar of the original operation. This mass had rapidly increased and was about 2 cm. in diameter. It was again excised together with a wedge-shaped piece of tongue wide of the edge of the growth. Microscopically the growth proved to be a small spindle-celled sarcoma. In answer to our enquiries as to the progress of this patient, Dr. Stern writes that in spite of all his efforts the child was taken from the hospital and soon died, apparently from recurrence of the growth.

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Albert. Wiener Mediz Presse, 1885, p. 171.

CASE 20.—Female, æt. 56. History of a year's difficulty in deglutition. Soon after this was first noticed she found a tumour on the tongue which slowly but progressively increased in size causing the difficulty in swallowing and rendering speech indistinct. On examination there was a smooth, rounded tumour at the posterior part of the tongue, extending up to the soft palate and hiding the isthmus of the fauces. The mass was well defined and involved the whole breadth of the tongue. It was moderately firm in consistence. April 1883 an incision was made from the chin to the hyoid bone and then along the great cornu of the bone on the right side. Opening up the buccal cavity, the whole tongue was excised. Two days after the operation patient developed pneumonia from which she died on the eighth day. The tumour under the microscope proved to be a round-celled sarcoma.

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Schulten. Finska Läkaresällskapets Handlingar, 1888, p. 689.

CASE 21.—Female, æt. 32. There was a tumour the size of a hen's egg embedded in the root of the tongue, projecting into the pharynx, reaching and pressing upon the posterior wall of that passage, so that it was with difficulty that even fluids could be swallowed. The mucous membrane over the tumour was healthy, and there was no enlargement of the cervical lymphatic glands. There was no pain in the tongue. Tracheotomy was performed and an incision made immediately about the hyoid bone. Both lingual arteries were ligatured and the whole tongue, with the embedded tumour, removed. The patient was fed by means of an œsophageal tube and recovery was rapid. She left the hospital after seven weeks. The tumour was found to be a small round-celled sarcoma. The patient was well and free from recurrence three years after the operation (Shambaugh).

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Scheier. Berlin Klinische Wochenschrift, 1892, p. 534.

CASE 22.—S. R., female, æt. 28. In June 1890 the patient had pain in tongue and slight difficulty in deglutition, and in September a small ulcer was found on the base with induration around. Mercurial inunction had no effect, and in January 1891 the difficulty in swallowing and the pain had increased. There was slight difficulty in breathing and limitation of protrusion of the tongue. The anterior part of the tongue was normal but a large tumour was found at the base, projecting into and reaching the posterior wall of the pharynx and obstructing a view of the epiglottis. There was some necrosis on the surface of the mass, which was of soft consistence. Below the angle of the jaw on the left side was a movable, enlarged gland. Patient was given iodides in large doses for five days without result. January

16th 1902 tracheotomy was performed and an incision carried from the left angle of the jaw to the hyoid bone. The lingual artery was tied and the jaw divided between the molar teeth. The whole tongue was removed and the cavity packed with zinc chloride and gauze. The growth was a sarcoma with small round-cells containing relatively large nuclei. Numerous thin-walled vessels were present and muscle-fibres were embedded in the new growth. Five weeks after the operation recurrence had taken place in the floor of the mouth for which another operation was performed. In April 1891 the glands of the left side of the neck became attacked by growth. These increased rapidly and those of the right side becoming affected tracheotomy had to be performed. Death occurred in December 1891. At the post-mortem examination a mass as big as an infant's head occupied the left side of the neck, extending as far downwards as the clavicle and crossing over to the right side anteriorly. At the level of the seventh rib and just to the right of the sternum was a small subcutaneous mass of growth. Secondary deposits were found in the lungs and in the peritoneum. At the base of the tongue was a mass of growth extending into the superior aperture of the larynx as far as the level of the right true vocal cord. Microscopically all these secondary growths had the same structure as the original tumour.

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Hutchinson. *Medic. Chir. Transact.*, 1885, vol. lxxviii., p. 311.

CASE 23.—Male, æt. 22. When ten years of age the patient first noticed a swelling of the left side of the tongue with a rough papillary mucous membrane over the posterior part. The tumour gradually increased in size until in seven years the tongue could not be protruded from the mouth and speech was difficult. There was then a rounded mass embedded in the tongue, leaving only the tip free and bulging the submaxillary space. The mucous membrane was healthy except for a coarse papillary growth over the posterior two-thirds of the left side, and except for this part it was movable with the superficial stratum of muscle over the tumour. No operation was performed for a year during which time the increase in volume of the tumour was considerable. In 1881 tracheotomy was performed. The tumour was found on exploratory incision to be non-encapsuled and, failing by simple methods to reach its posterior limit, Mr. Hutchinson divided the jaw and removed the whole tongue by the *écraseur*. Recovery was good. The tumour was spherical, measuring two and a half inches across, and replaced the muscle substance of the tongue except in the anterior part. The anterior part of the mass was softer and very vascular and inseparable from the surrounding muscle. It was examined by Eve, Klein, and Godlee, who pronounced it a round-celled sarcoma with large nuclei and ill-defined cell substance. The muscle fibres were atrophied and there was a tendency to the formation of fibrous tissue. The patient went on well for some time but ultimately recurrence took place *in situ* and he died two and a half years after operation. No post-mortem examination was obtained.

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Meyer. *New York Med. Journal*, 1892, vol. lv., p. 133.

CASE 24.—A patient was shown at the New York Surgical Society in October 1891 in whom a growth involved not only the posterior part of the tongue but the right tonsil and the adjacent pharyngeal wall. It caused great pain. The lymphatic glands of the right side of the neck were

enlarged. Tracheotomy was performed and the right external carotid artery was ligatured. The whole of the tongue and a part of the pharyngeal wall on the right side, together with the enlarged lymphatic glands, were removed by an incision in the neck. The patient was fed by means of an œsophageal tube and did well. The report mentions that the enlarged glands were sarcomatous but gives no report of the histological examination. In reply to our enquiries Dr. Meyer writes—"The tying of the external carotid and the removal of the ascending ramus of the inferior maxillary bone proved to be a great comfort during the operation. By opening the œsophagus at the level of the cricoid cartilage I had complete access to the parts involved and could do a clean extirpation in continuity under the guidance of my eyes. The patient continued to do well without any symptom of local recurrence for more than one and a half years. Then he lost his job and, in a fit of melancholia, he shot himself through the temple, dying instantly."

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Shambaugh. *American Journ. of Med. Sci.*, Jan., 1902.

CASE 25.—A. S., male, æt. 38. Family history good. In September 1900 he was cachectic with sunken face and sallow skin. For eight months he had had slight dysphagia which increased to such an extent that he was driven to using his finger to push his food past the swelling, whilst later nothing but fluids could be taken. His voice had become much altered and his respiration was embarrassed. There were no enlarged glands in the neck. Inspection of the oral cavity showed the anterior part of the tongue to be normal. The posterior part of the mouth was occupied by a tumour the size of a hen's egg, lodged in the left side of the tongue and bulging upwards the dorsum so as to hide from view the pillars of the fauces and the soft palate of that side. Its surface was covered by thin, smooth mucous membrane containing dilated venules in places. The mass felt soft and appeared to be attached deeply in the root of the tongue. Embedded in the right side of the root of the tongue was an extension of the growth, covered by smooth mucous membrane, and extending as far forwards as the circumvallate papillæ. During the following fortnight the patient had an attack of severe hæmorrhage from his throat, and a week later a small piece of the tumour was removed for microscopic examination. It proved to be a sarcoma made up of large spindle-shaped cells arranged in fascicles and separated by some fibrous intercellular substance. There were numerous blood vessels and some hæmorrhagic patches. The patient was not operated upon and was lost sight of, so no further history is available.

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Fiedler. *Ztschr. f. Med. Chir. und Geburtsch Leipzig*, 1864, vol. iii., p. 305.

CASE 26.—Male, æt. 40. There was a tumour growing rapidly in the base of the tongue, projecting backwards into the pharynx, causing difficulty in deglutition and in respiration. There was pain on movement of the tongue, but the condition of the mucous membrane over the tumour is not stated. The lymphatic glands in the neck were enlarged. The whole tongue was extirpated by the galvano-cautery, but rapid recurrence of the growth took place in the stump and the patient died of exhaustion. The growth on section was found to be a spindle-celled sarcoma.

Eva. Pathological Soc. Trans., 1886, vol. xxxvii., p. 223.

CASE 27.—This and case 29 form the reports of two specimens of lingual tumour preserved in the Museum of the Royal College of Surgeons and are unfortunately devoid of any clinical history. A small firm tumour of uniform consistence and pale in colour removed from the tongue. Microscopically it consisted of a loose fibrillar network filled with oval and fusiform cells. No muscle fibres were seen. From the abundance of cellular elements present this tumour was pronounced to be a fusiform celled fibro sarcoma.

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Perkins. Annals of Surg., 1896, vol. xxiii., p. 585.

CASE 28.—P. J., male, æt. 26. At the age of four the patient had a small tumour on the upper surface of the tongue. This gradually increased in size until at the age of twelve years it was the size of a walnut, embedded in the tongue and bulging on both the superior and inferior surfaces. From this time up to twenty-five years it remained stationary, causing no discomfort, but it then began to increase rapidly in size, especially in the anterior extremity, and in October 1892 it was removed locally; in twelve months there was an active recurrence in situ, enlarging to such an extent as to interfere with deglutition and respiration and causing bilateral dislocation of the jaw when the mouth was opened. The growth was confined to the anterior three-fifths of the tongue, symmetrically placed, smooth, firm and elastic. The mucous membrane was everywhere healthy except at the scar of the former operation. There were no glands enlarged in the neck. The tongue was removed three-quarters of an inch behind the growth by Whitehead's operation and the patient did well. A vertical section of the part removed showed that the muscular part of the tongue was almost entirely replaced by growth, which was very firm and greyish-white in colour. Microscopically it was found to be a small round-celled fibro-sarcoma. The patient went on well until September 1895 (twenty months after the last operation) when there was again recurrence in the stump of the tongue; there was no ulceration of the mucous membrane. The lymphatic glands in both submaxillary regions were enlarged. The tumour caused no pain or inconvenience and the patient declined to undergo any further operations. Dr. Perkins writes that, though he did not see the patient himself again after the last mentioned date, he heard indirectly that the patient died "some two or three years after the operation was done," apparently from extension of the recurrent growth.

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Eva. Trans. Path. Soc., 1886, vol. xxxvii., p. 223.

CASE 29.—In the left side of the base of the tongue was a firm, rounded tumour, covered with normal mucous membrane. The base of the tumour was indistinguishable from the healthy muscular substance surrounding it. The mucous membrane of the left side of the soft palate and epiglottis was oedematous. Microscopically the tumour consisted of masses of clear, round cells contained in a delicate fibrillar network. There were numerous dilated lymphatic sinuses, especially in the deep layers of the mucosa. The tumour was a round-celled sarcoma probably arising from a connective tissue hyperplasia secondary to lymphangiectasis, which ultimately underwent malignant changes.

**Adeno-sarcoma of the tongue.**

Godlee. *Trans. Path. Soc.*, vol. xxxviii., p. 346.

CASE 30.—This case, though strictly speaking a sarcoma of the tongue, is not included in the foregoing account of the sarcomata arising in the connective tissue substance of the tongue, but it is placed in a separate category owing to its probable origin in the small mucous gland, described by Blaudin and Nuhn, situated in the side of the tongue deep to the fibres of the styloglossus muscle. Female, *æt.* 24.—On the left side of the tongue, near the tip, was an ulcerated surface on the summit of an elevation which, when grasped, was firm to the touch and of the size of a hazel-nut. On exploratory puncture the needle struck in the centre of the growth a hard mass which proved to be a salivary calculus. The whole tumour was eventually removed. Microscopic examination showed it to be an adeno-sarcoma consisting of irregular shaped acini of glandular elements of various sizes with large nucleated cells; some of the acini were distended into small cysts. There were numerous blood-vessels present. There was no recurrence in three years.

**Secondary sarcoma of the tongue.**

James, G. B. *Trans. Path. Soc.*, 1898, vol. xlix., p. 91.

CASE 31.—There was a mass embedded in the substance of the tongue, secondary to a growth almost encircling the lower four inches of the *œsophagus*. Microscopical examination of the primary growth showed large alveoli packed with spindle-shaped cells and the mass in the tongue presented similar characters.

Michael. *Handbuch der Laryngol und Rhinol.* Heimann, 1899, vol. ii., p. 634.

CASE 32.—This case has been pronounced to be a secondary sarcoma of the tongue but, from the report, the lingual tumour seems to be in direct continuity with a recurrent mass of sarcoma in the tonsil rather than a secondary metastatic deposit. Patient, *æt.* 30, had a sarcoma of the tonsil incompletely removed by a tonsillotome, and when seen some weeks later a large recurrent mass was found. Deglutition was impeded and speech difficult. Protrusion of the tongue was impossible. The cervical lymphatic glands were swollen to the size of the fist. A small piece removed by a sharp spoon proved on examination to be sarcomatous but the wide extension of the disease precluded any operation. The patient soon became cachectic and died. Post-mortem.—The tumour had involved the pharyngeal wall and the posterior half of the tongue. No metastases were found.

Gerster. *New York Surg. Soc.*, 1894.

CASE 33.—During the discussion following the exhibition of Abbe's case of sarcoma of the tongue Gerster spoke of having operated upon a case of this disease, and he has been good enough to send us a report of the case extracted from the Mount Sinai Hospital records. L. B., male, *æt.* 48. No specific history. Right tonsil removed a year before. The patient first noticed a swelling in the left tonsil four months previously, and two months later a swelling in the left side of the neck below the jaw. The left tonsil was enlarged by a hard, waxy-looking mass, which extended to the soft palate, side of tongue, and pharyngeal wall. The infra-maxillary glands were enlarged

and hard. November 21st 1893, the tongue and infiltrated tissues were removed by Langenbeck's operation after preliminary tracheotomy. The patient died five days later from pneumonia. This case is one of extensive sarcoma of the tonsil with direct invasion of the side of the tongue, and therefore cannot be included in our list of primary lingual sarcomata.

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*The following cases have been recorded in the various periodicals under the heading of "Sarcoma of the Tongue," but they are not included in compiling the foregoing report owing to the doubt as to their true nature or from the unsatisfactory nature of the report. Enquiries of the respective authors have failed to elicit any replies to verify the diagnosis of sarcoma. We append at the foot of each abstract our reason for their exclusion.*

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Jacobi. Americ. Journ. of Obstetrics, May, 1869, p. 81.

CASE 34.—Tumour of the dorsum of the tongue, congenital, the size of a hazel-nut at birth, but increasing to the size of a walnut in three months. It was firm, elastic, rounded and deeply furrowed on its surface, covered by a large network of capillaries and bright-red in colour. It was removed by the cautery and found to contain round and spindle-shaped cells. The child was quite well when seen again five months later. This tumour might have been composed of embryonic tissue, and Butlin gives it as his opinion that the case might have been one of angioma or fibroid nævus.

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Hüter. Berlin Klin. Wochensch, 1869, p. 346.

CASE 35.—Only a brief note of this case can be obtained. A tumour was present in the mid part of the dorsum of the tongue in a young woman who, at the time, was five months pregnant. The increase in the size of the growth was rapid and in two months it was removed. There is no account of the macroscopic or microscopic examination of the tumour. No further history of the patient is given, nor is the possibility of its being a gumma excluded.

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Mikulicz et Michelson. Atlas der Krank. der mund. und Rach. Tafel xxxv.

CASE 36.—A. P., female, æt. 24. Three months' history of a small tumour on the dorsum of the tongue. Gradual increase in size during that time with bleeding on mastication. It was pedunculated, measured 1·5 cm. in diameter, and was firm in consistence. There was no induration at the point of attachment of the pedicle to the tongue, and there was nothing to suggest any continuity of the growth through the pedicle into the lingual substance. It was cut off and the wound cauterised. There is no further note on this case either of clinical history or of a microscopical examination of the tumour, but the case is recorded as a fusiform-celled sarcoma. The report is too meagre however to justify its inclusion in a list of true sarcomata of the connective tissue of the tongue.

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Mercier. Rev. Med. de la Suisse Romande, 1890.

CASE 37.—J. B., male, æt. 36. Tumour on the dorsum of the tongue which had been slowly growing for eight years, but for four months before removal had remained of the same size. It was pedunculated, flattened on

the surface from contact with the palate, and covered by hypertrophied mucous membrane. In consistence it was firm and it measured  $2\frac{1}{2}$  cm. in diameter. It was removed with a wedge-shaped piece of the tongue and examined, when it was found to consist of spindle-shaped cells, whilst in the centre of the growth well-marked fatty degeneration had taken place. No further history of the case is given. This case is probably one of pedunculated fibroma of the tongue.

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Targett. Guy's Surgical Reports, 1873.

CASE 38.—C. J., æt. 10 months. Three months previously the mother noticed a small sore under the tongue. This sore bled occasionally, it increased, and a lump developed under it. When admitted under Mr. Howse, there was a rounded ulcer on the inferior surface of the tip of the tongue, central in position and touching the two lower central incisors on protrusion of the organ. The edges were raised and rounded, the surface flat and spongy-looking. It was removed by the cautery, and Mr. Howse expressed the opinion that sarcomatous tissue was present in the base on examining the mass in the fresh state. There is no mention in the report of a microscopic examination. Nothing further is known about the child, but the case is probably one of an irritative tumour due to contact with the sharp incisor teeth and known on the Continent as Riga's disease.

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Onodi. *Revue de Laryngol*, October, 1893.

CASE 39.—Female, æt. 17. History of eight months' duration of a small tumour on the left side of the base of the tongue, with gradual increase in size, elastic, vascular, painless, and with a clearly defined margin. A small piece was removed for microscopic examination, and was pronounced to be a fibrosarcoma. The patient refused to undergo any radical operation and when seen a month later the condition was unaltered, the hollow remaining from which the piece had been removed. Butlin remarks: "It is not a character of sarcoma to remain stationary after a piece has been punched out of it, and the case accords with hypertrophy of a lymph follicle."

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Perman. *Buffalo Med. and Surg. Jour.*, 1894, p. 148.

CASE 40.—V. E., female, æt. 30. For some six months the patient had noticed streaks of blood in the saliva, and later some slight difficulty in deglutition. On examination by means of a laryngoscope a small tumour was found at the base of the tongue with rugose surface, placed chiefly on the right side, but crossing the middle line and extending down as far as the epiglottis. It was soft to the touch and there was no induration of the surrounding parts. The patient was given iodides for a week with no effect in diminishing the size of the tumour, and a piece of the growth was then removed for microscopical examination. It was reported as sarcomatous (variety not stated) but the patient's husband refused to allow any further operation. Accordingly the tongue at the side of the tumour was injected every few days with two grams of a 1:1500 solution of pyocetanin, followed on two occasions by removal of small pieces of the growth with a snare. In three months the swelling had gone down, and in twelve months there was no signs of recurrence. It seems extremely unlikely that this case could have been one of sarcoma; probably it is an instance of hypertrophy of the lymphoid tissue at the base of the tongue.



Barker. Holmes' Syst. of Surg., vol. ii., p. 576.

CASE 41.—This patient was under Godlee for a tumour situate on the dorsum of the tongue. It was pendulous and soon recurred after removal. Subsequently several growths of a similar kind appeared on the skin in various parts of the body. Barker suggests that the case was one of multiple sarcoma, one growth appearing on the tongue.

Bloodgood. Johns Hopkins Hosp. Bulletin, 1894.

CASE 42.—Patient, æt. 46. History of a tumour for nine months growing in the right side of the inferior aspect of the tongue and extending into the floor of the mouth. A few weeks earlier the tumour had been punctured under the supposition that it was a ranula, and the growth rapidly fungated, forming a mass lightly wedged in between the tongue and the teeth and covered with a foul discharge. A piece was removed and found to be a mixed-celled sarcoma. Tracheotomy was performed and Professor Halstead removed the right side of the tongue, the floor of the mouth, and a part of the body of the lower jaw, together with the glands of the neck. Dr. Bloodgood writes that the tumour seemed to spring from the floor of the mouth. It extended from the symphysis of the jaw to the angle and infiltrated and involved the periosteum of the jaw, but the bone was not involved. It infiltrated beneath and only slightly into the tongue. It was difficult to say whether it originated from the connective tissue in the floor of the mouth or from the periosteum of the lower jaw. The lymphatic glands of the neck showed no enlargement. There was no recurrence of the growth in eight years. This case cannot obviously be included with those of primary sarcoma of the tongue.

Bleything. New York Med. Journal, 1888, vol. xlvii., p. 683.

CASE 43.—Male, æt. 17, who had an ulcer on the right margin of the tongue, half an inch from the tip. It was covered by granulations; the base was indurated and under treatment by cauterisation it increased in size. The ulcer was excised, and on examination showed "areas of grouped cells with new areas of infection around gorged vessels." Six years later the tongue was quite healthy. The ulcer was declared to be sarcomatous but it seems to us more probable that it was due to chronic traumatism and the microscopic appearance to septic infection.

Melchior-Robert. Rev. du Chir., 1899, vol. xix, p. 545.

CASE 44.—S. T., female, æt. 64. Spherical tumour noticed for three months on the right side of the tongue, 3 cm. behind tip. It was pedunculated, of firm consistence, bleeding slightly on mastication. The mucous membrane over it was ulcerated by contact with the teeth. There was no induration in the tongue at site of implantation of the pedicle. The tumour was removed and on examination was said to consist of a fibrillar connective tissue enclosing fusiform cells with large nuclei and to present signs of septic infection towards the surface. There is no mention of the microscopic structure in the pedicle or in the subjacent area of the lingual tissue, and no history is given of the patient subsequent to the operation. The case is probably one of pedunculated fibroma of the tongue.

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## Tongue accepted

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# A RESEARCH UPON THE METABOLISM OF A PATIENT SUFFERING FROM DIABETES INSIPIDUS, FOLLOWING UPON FRACTURE OF THE SKULL.

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AND

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During their tenure of the Beanev Scholarship (Guy's) and of the Gillson Scholarship (Society of Apothecaries).

(From the Physiology Laboratory, Guy's Hospital.)

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WILLIAM T., æt. 44, an Insurance Agent, living in Berkshire, was a married man, but had no children. He neither smoked nor drank alcohol. He had been perfectly well until, at the beginning of June, 1901, he met with a bicycling accident. He did not remember the nature of the accident, beyond the fact that he was "shot over the handle bars." He was found unconscious, and remained so for a fortnight; during which time his medical attendant, Dr. Loveday, collected and examined quantities of cerebro-spinal fluid which dripped from his nose.

On recovering consciousness, he suffered from extreme thirst, and drank weak lemonade incessantly. At that time Dr. Loveday found he was passing from two to three gallons of urine a day, of a specific gravity 1000.

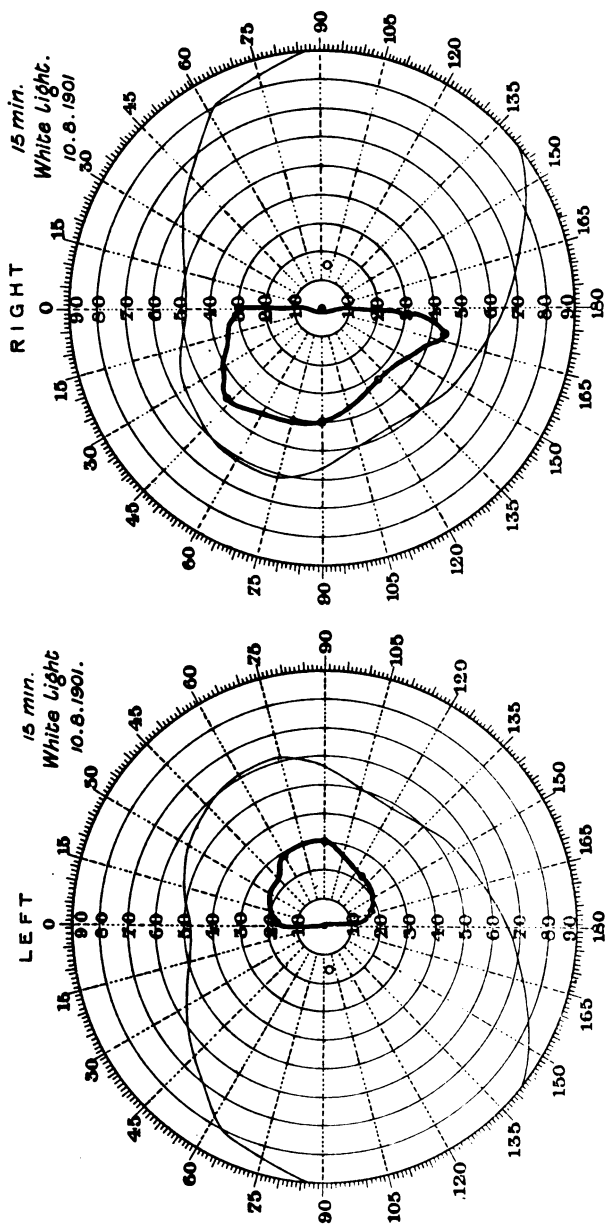
On August 6th, 1901, he came to Guy's Hospital, and was admitted under Dr. Hale White. He was a talkative, healthy-looking man, with a good, but not voracious, appetite, and extreme thirst. He walked about the ward, but with some difficulty owing to affection of his sight. His eyes had previously been perfect, so far as he knew; but there was now internal strabismus of the left eye, and inability to move it out beyond the middle line. There was partial ptosis of the left eyelid. There was no exophthalmos. His pupils reacted well to accommodation and to light. There was temporal hemianopsia, as depicted in the following charts, the fixation point being involved in the left eye but not in the right.

Vision of the left eye was  $\frac{4}{60}$ ; of the right  $\frac{6}{12}$ . He could read J 1 slowly. The discs looked pale, with large physiological caps. Probably some atrophy was present. Later the optic atrophy became very marked, the visual fields still more contracted.

His knee-jerks were sluggish, but otherwise his nervous system appeared normal. He could not hear well with his left ear, but this he said was also the case long before the accident. His respiratory, cardiac, and alimentary systems seemed perfectly sound.

The diagnosis was that he had fractured the base of his skull; that the fracture included the basisphenoid; that callus was probably affecting his optic chiasma; and that, as a result of the head injury, he had hydruria or diabetes insipidus.

The thirst and the passage of large quantities of urine were the all-prevailing symptoms. He was not excessively hungry, but he could not refrain from drinking water or lemonade, at short intervals, both day and night. At this time the urine was measured in the ward every day, and he passed from 3150 to 3210 daily. The specific gravity varied from 1002 to 1006, but was usually 1004. The reaction was acid; no albumen or sugar were present; and the urea, roughly measured in a Southall's ureometer varied from 0.4 per cent. to 0.7 per cent. During August, 1901, he was given potatoes with every meal, and glucose 3j. three times a day. On two occasions, the ward clerk states that sugar was present in the urine, namely, on



August 26th 34.6 grains; and on August 27th 25.4 grains; the estimation was made by Fehling's solution; but no confirmation of the reducing body being sugar was made, either by fermentation with yeast, or by the phenyl hydrazine test. In view of later investigations, it seems doubtful if sugar were ever present.

The patient's temperature was normal, or just subnormal throughout.

On August 17th, he was supplied with spectacles, which enabled him to read fair-sized type readily.

#### THE OBJECTS OF THE RESEARCH.

Atwater and Langworthy (1) have recorded no investigation of metabolism in diabetes insipidus up to 1896; since then, one paper only, by Strubell (22), has been published on the subject; it was proposed, therefore, to investigate the following points:—

1. Whether the excretion of urea differed in any way from that of a normal person.
2. Whether the excretion of uric acid were altered.
3. Whether or not the patient's carbohydrate assimilating power were defective; that is to say, whether, by giving as much carbohydrate of different kinds as a healthy person can take, glycosuria could be produced.
4. What relation the hydruria bore to the general blood-pressure, and to the specific gravity of the blood.
5. What proportion of the fluid consumed was recovered in the urine.

For this purpose the patient was transferred to the private room in Stephen ward on September 9th, 1901, and two special nurses, whose sole duty was the care of the patient, were appointed to look after him. For the first three days he was put upon a preparatory diet of milk. The experiment began upon September 11th, and continued without interruption until October 15th.



During the whole of this time his only medicine was the following mixture :—

R	Ext. Nuc. Vom. liq.	...	...	℥j.
	Tr. Gent. cum Glyc.	...	...	ʒj.
	Acidi Nitrohydrochlor. dil	...	...	℥x.
	Aq ad	...	...	ʒj. t.d.s.

For the first five days he also had Rubinat water ʒij. o.m., but after September 17th this was omitted.

#### THE METHODS USED.

The patient was kept in bed during the morning, but used to get up in the afternoon from 2 p.m. till 7 p.m. He took no vigorous exercise, but once or twice he was walking out of doors. Exercise seemed not to influence that part of his metabolism upon which research was being made. If glycosuria had been produced, it was intended to see if increased exercise gave a better assimilating power for sugar, but he always assimilated all the carbohydrate he could eat.

The precautions used in accurately weighing his food, administering it, and collecting his urine and fæces, were the same as those adopted in a previous research, and are described in the Guy's Hospital Reports, vol. lvi., p. 56. The patient was weighed daily, the weight of his clothes being deducted. The food consisted of milk alone for the first seven days. The milk was supplied in sterilised quart bottles, twenty gallons at a time, from the same churn, by the Aylesbury Dairy Company; and this, being all of the same composition, led to a great saving in time and labour. Afterwards, as will be seen in the diet table (Table V.), biscuits, butter, Liebig's meat extract, eggs, arrowroot, cane sugar, and grape sugar were added. Unlimited water, flavoured with essence of lemon and sugar, was allowed; but it was accurately measured; twenty ounces were given at a time, and the patient drank from the measuring glass, as he required it, until the latter was empty, when a fresh twenty ounces were prepared.

The nitrogen in all the food stuffs was directly estimated by Kjeldahl. The carbohydrate, and the fat, were calculated from diet tables (5).

Each day, the urine of the previous twenty-four hours was measured in graduated cylinders. The total nitrogen in it was estimated by Kjeldahl; the urea, by the Mörner-Sjöquist (10) method; the uric acid, by the Hopkins (8) potassium permanganate process; and the specific gravity by means of a specific gravity bottle. The nitrogen in the fæces was determined by the process described in the Guy's Hospital Reports, vol. lvi., p. 58.

In the same paper will be found tables and full references for figures obtained by different observers during the last twenty years upon the following points in healthy individuals:—

1. The proportion of the total nitrogen in the urine which is in the form of urea.
2. The relation of uric acid to urea.
3. The relation of uric acid nitrogen to the total nitrogen in the urine.
4. The daily amount of nitrogen in the fæces.

It is therefore unnecessary to repeat them. In the appendix are given full tables of the analyses made during the present research.

#### THE PROTEID METABOLISM.

It will be seen that the patient's nitrogen metabolism was that of a normal person. The urine passed daily was, upon the average of the whole period of five weeks, 7400 c.c., whereas the normal quantity is 1500 c.c. The specific gravity was 1004, with slight variations above and below this. There was, therefore, a very large increase in the amount of water eliminated by the kidneys; but there was neither an increase nor a decrease, nor an altered proportion, in the quantity of urea. Over the entire period the average daily excretion of nitrogen as urea was 14·2 grams, equivalent to 30·4 grams of urea. The maximum was 18 grams of urea nitrogen, or 38·6 grams of urea, in one day; the minimum, 11 grams of urea nitrogen, or 23·6 grams of urea. Over the whole period, of every 100 grams of urine nitrogen, 88·5 grams were in the form of urea. The variations were between a minimum of 79·6 per cent. and

a maximum of 95·5 per cent.; and these are well within limits found in healthy persons, in whom the average is about 86 per cent. (4).

Parkes (11) supported the view that diabetes insipidus was found in three forms:—

1. Cases where there was no increase or decrease of tissue metamorphosis.

2. Cases where there is a decided decrease of tissue metamorphosis.

3. Cases where there is evidence of increased tissue metamorphosis, as shown by the increase of some of the urinary solids.

Ralfe (12) gives the term *hydruria* to those cases in which the aqueous superflux is most marked; reserving the term *polyuria* for instances of persistent drain of one or more of the solid constituents of the urine. The present patient's condition appears to have been one of simple hydruria.

This being so, it is interesting to examine his excretion of uric acid. It has been urged against the Hopkins permanganate process, that it is inapplicable to very dilute urines. This is not so. It is true that the correction figure, often as much as ·002 gram, is relatively large when the total uric acid in 100 c.c. is only ·015 gram, as is frequently the case with so watery a solution; but a similar correction is necessary in other methods; and, as will be seen by referring to Table III., the uric acid results closely correspond with those of healthy individuals. With so much fluid to dissolve the uric acid, it might be expected that a larger quantity than usual would have been excreted. Over the whole period of five weeks the average daily excretion of nitrogen as uric acid was 0·286 gram, representing 0·86 gram of uric acid per diem. This is somewhat higher than 0·55 gram, the average given by Schäfer (16) for health; but is within the limits of healthy variation (see Guy's Hospital Reports, vol. lvi., p. 70). Moreover, the relation of the uric acid to the urea was similar to that of normal persons. Taking the average for the whole five weeks:—

$$\frac{\text{the uric acid}}{\text{the urea}} = \frac{1}{3\frac{1}{2}}$$

Examination of Table III., however, brings out an interesting point. For the first seven days, the patient was taking milk only; during this time the uric acid was very low, the average for the seven days showing that:—

$$\frac{\text{the uric acid}}{\text{the urea}} = 1\frac{1}{4}$$

the variations being from  $\frac{1}{228}$  to  $\frac{1}{132}$ . It is well known that, upon milk diet, the uric acid secretion is thus small. Upon September 19th, the first day that his diet was increased by the addition of breakfast biscuits, butter, and two eggs, the ratio rose to  $\frac{1}{87}$ . So rapid a rise was unexpected; the great increase was looked for upon the next day (September 20th) when Liebig's extract of meat was also given, because it has been found by Strauss (21) and by Smith Jerome (19) that meat extracts give a high ratio  $\frac{\text{uric acid}}{\text{urea}}$ .

With Liebig in the diet, the figure was once as high as  $\frac{1}{18}$  (September 30th); but it was a surprise to find that, without meat extract, and only biscuits, butter and two eggs in addition to the milk, the ratio suddenly rose from  $\frac{1}{177}$  to  $\frac{1}{87}$ . It would point to the importance of giving nothing but milk if the production of uric acid in the body is to be rapidly diminished.

The average ratio  $\frac{\text{uric acid}}{\text{urea}}$  for the period when other foods besides milk were given was  $\frac{1}{29}$ ; and during this period he had milk and eggs but no butcher's meat of any kind. The limits were from  $\frac{1}{18}$  to  $\frac{1}{80}$ . Normal persons show even wider variations than these (loc. cit., p. 70).

The nitrogen in the fæces was estimated daily in order to obtain a balance between the intake and the output of nitrogen. The results will be found in Table II., and may be of use to others; not many estimations of fæces nitrogen, continuous for weeks, have been published. In the present instance, though there was no active diarrhœa, the average daily nitrogen in the fæces was 2.35 grams, constituting 12.75 per cent. of the total nitrogen excretion, and these are higher figures than are usual. Most persons thus excrete from 0.5 to 2.0 grams per diem (for

references and comparative figures see Guy's Hospital Reports, vol. lvi., pp. 77 and 78).

During the five weeks' investigation the nitrogen taken in the patient's food exceeded that recovered in the urine and fæces by 76·41 grams, an average deficit of 2·25 grams per diem. At the same time, however, the patient continuously increased in weight. (See Table IV.). He was 4083 grams heavier at the end of the research than at the beginning. This increase in weight was no doubt partly fat, but part of it may have been proteid, and for every 100 grams of proteid tissue built up, 3·3 grams of nitrogen are required. Therefore the building up of 4083 grams of proteid would require a retention in the body of 135 grams of nitrogen. Thus, if half the increased body weight were due to proteid, more than all the nitrogen deficit would be accounted for.

The fluid taken by the mouth, and the urine excreted, having been carefully measured, and the water in the solid food calculated, it is of interest to see what proportion of the fluid consumed was recovered in the urine. Table I. shows that upon five occasions only did the urine exceed in volume the fluid taken by the patient as water, milk, medicine, and solid food. Two such occasions were upon the last two days, when the patient with difficulty restricted his drinking to  $3\frac{1}{2}$  litres per diem. Over the entire five weeks, an average of 90·87 per cent. of the fluid consumed was recovered in the urine, the least amount being 78 per cent. Little research upon this subject seems to have been made in normal people. The weather at the time was warm, but the patient did not visibly perspire; 90 per cent. of the total intake of fluid is probably more than is excreted in the urine by healthy persons, but perhaps so high a figure might be expected when such quantities were drunk. In this estimate, no allowance has been made for the water formed in the body from the hydrogen in the food, but the tables afford all the necessary data should such a correction be desired.

#### THE CARBOHYDRATE ASSIMILATING POWER.

The pathology of diabetes insipidus is little known, though it will be mentioned again presently. In most cases no immediate

cause for the onset can be assigned. Nervous inheritance, fright, or emotion predispose to it sometimes. In such instances, perhaps, there is little reason to expect any relation between it and glycosuria. When, however, a head injury is the exciting cause, it is thought that a centre in the medulla oblongata has been in some way affected. Eckhardt (7) showed that hydruria could be produced by chemical or mechanical stimulation of the superior vermis of the cerebellum, and Claude Bernard proved by needle puncture that there were two points close together in the medulla, injury to one of which led to glycosuria, to the other, hydruria. It would not be remarkable if so gross a lesion as a fracture to the base of the skull should interfere with both these centres. The present writers expected that, in diabetes insipidus of traumatic origin, the power of assimilating carbohydrate would be defective, even though in other forms of the disease this might not be the case. The result, however, was otherwise. Upon no single occasion could even a trace of sugar be detected in the patient's urine. Upon the sixteenth day of the research, there was added to his biscuits, eggs, Liebig and milk, as much cane sugar, grape sugar, and arrowroot starch in varying proportions as he could be persuaded to take. These forms of carbohydrate were chosen, rather than potatoes, for example, because, being dry, they could be more easily and accurately weighed. It is true that the amount he took did not often largely exceed that taken by a healthy man. Table VI. shows how much he consumed. Upon one occasion this reached 738 grams of dry carbohydrate in addition to proteid and fat, and several times he took over 500 grams in the twenty-four hours; he could not be persuaded to eat more. However, even these quantities, if not excessive, are at least equal to those consumed by healthy persons. He readily assimilated them, gained in weight, and never passed any sugar in his urine. His sugar assimilating power seems to have been very good. That this is the case in ordinary diabetes insipidus is well known, but evidence of interference with the "sugar point" had been expected when a severe injury had apparently so much disturbed the adjacent centre. It had seemed possible that the pathology

of traumatic and of non-traumatic diabetes insipidus might differ in this respect, but in the present instance this was not found to be the case.

#### THE RELATION OF THE HYDRURIA TO THE BLOOD.

It is conceivable that a cerebral injury might so affect the brain, that some portion of it, concerned in the subjective sensation "Thirst," was interfered with, and that, as a result of a constant and perverted sensation of thirst the injured person might be continually drinking to quench this subjective thirst; the hydruria then being the result of drinking, instead of the drinking a consequence of the hydruria. To determine whether or not this were the case, the specific gravity of the blood was investigated. If a subjective thirst were primary, the specific gravity of the blood would probably be low, if hydruria, high.

The chloroform and benzene method was adopted. The red and white corpuscles were counted by the Thoma-Zeiss hæmocytemeter, the hæmoglobin estimated both by Gowers' and by Haldane's hæmoglobinometers, and several readings were taken in each case. The results were as follows:—

Date and time.	Specific gravity of patient's blood.	Control (H.S.F.).
September 27th, 6.30 p.m.—		
No food since tea at 4 p.m. ...	1060 ...	1056
October 9th, 3 p.m.—		
No food since lunch at 12.30 p.m. ...	1061 ...	1056
October 12th, 3 p.m.—		
No food since lunch at 12.30 p.m. ...	1060 ..	1056

At these times the patient was having as much water to drink as he wished. In order to see the effect of restricting the fluid ingested, his blood was fully examined on October 12th, at 3.30 p.m., three hours after a meal, whilst water was still unlimited. The results were as follows:—

Specific gravity ...	1060.
Red corpuscles ...	5,800,000 per c.mm.
White corpuscles...	7969 per c.mm.
Hæmoglobin ...	102 per cent. (Gowers'). 108 per cent. (Haldane's).

On October 13th, at 10 a.m., he agreed to restrict his drinking as much as he possibly could. It was hard to do, but he was an excellent patient, and bore his thirst. Even now, however, he was drinking more than an ordinary man does (see Table I.). At 3 p.m., when he had drunk only one and a half pints since 10 a.m., the following readings were obtained :—

Specific gravity ...	1061.
Red corpuscles ...	5,550,000 per c.mm.
White corpuscles. .	7344 per c.mm.
Hæmoglobin ...	100 per cent. (Gowers'). 110 per cent. (Haldane's).

On October 14th, at 8 p.m., when he had excreted in the previous twenty-four hours 572 c.c. more fluid than he had taken, the results were little changed from those of the previous day :—

Specific gravity ...	1060·5.
Red corpuscles ...	5,550,000 per c.mm.
White corpuscles...	10,000 per c.mm.
Hæmoglobin ...	(too bad a light for Gowers'). 112 per cent. (Haldane's).

On October 15th, at 10 a.m., after another twenty-four hours restriction to three litres, and a passage of a further 223 c.c. more than was consumed, the patient's thirst was very great; and the readings were as follows :—

Specific gravity ...	1066 (several readings).
Red corpuscles ...	6,300,000 per c.mm.
White corpuscles...	7656 per c.mm.
Hæmoglobin ...	112 per cent. (Gowers'). 122 per cent. (Haldane's).

It will thus be seen that, even with unlimited fluid, the patient's blood was a little more concentrated than normal; the specific gravity, the red corpuscles, and the hæmoglobin all gave slightly higher readings than do those of an ordinary man; after two days during which the fluid was, in spite of thirst, kept down to three litres per diem, the concentration of the blood was considerably increased, as shown by the rise in the specific gravity, in the number of red corpuscles per c.mm., and in the



percentage of hæmoglobin. It should follow from this that the hydruria was primary, and the thirst secondary; in order to prevent dehydration of his tissues the patient was obliged to drink continually. Probably the blood change was less marked on the first than on the second day of restriction, because the tissues were able to give up water to the blood during the first twenty-four hours, but were unable to continue the supply for forty-eight hours.

It would be interesting to investigate the relation between the hydruria of granular kidney and the specific gravity of the blood in a similar way.

An attempt was also made to discover whether the patient's blood-pressure was in any way different to normal, in case the hydruria might be in some way connected with increased general blood-pressure. Oliver's blood-pressure gauge, with a fluid pad, was used. With this instrument it is difficult to be more than approximate in the readings. Moreover, the readings obtained cannot be regarded as more than of relative value; they serve for comparison with others made with the same instrument. Table VII. shows that, when lying in bed with his arm horizontal, the radial blood-pressure was, throughout, what the instrument recorded as from 135 to 150 mm. Hg., with an average of about 140 mm. Hg.; and when the patient was standing up, with his arm supported on a level with his heart, the pressure recorded on the radial varied on different occasions from 160 to 180 mm. Hg. These figures cannot be regarded as absolute, but only relative; they are high, and yet, when similar observations were made upon seven different healthy hospital students, the readings were all about 140 mm. Hg. for the lying position, and from 170 to 180 mm. Hg. for the standing. Hence, since the readings both for the patient and for healthy students were almost identical, the conclusion is that the instrument in each case gave too high a figure, and that the patient's general blood-pressure was not very different from that of a young and healthy student.

## OTHER CASES OF DIABETES INSIPIDUS IN GUY'S HOSPITAL.

Hydruria of traumatic origin is not of common occurrence. Between the years 1883 and 1902 no such case was in the wards of Guy's Hospital. During these twenty years, 11 cases of diabetes insipidus were admitted. Though none were traumatic, the following abstracts from their medical reports are inserted here, because they have not been previously recorded:—

CASE 1.—Walter M., æt. 21 (Vol. 100, No. 262), was in the hospital from August 10th to September 13th, 1887. His family history seemed to have no bearing on his condition. He had begun to suffer from hydruria and extreme thirst two months before admission. He also suffered from headache and sleeplessness, and his appetite was poor; he could assign no cause for his symptoms, and had had no injury. His temperature was frequently as high as 108° F., and for this no explanation was found. His urine varied from 100 to 170 ounces per diem, with a specific gravity 1000 to 1006. It was acid in reaction, and contained from 300 to 500 grains of urea in the twenty-four hours. It never contained albumen or sugar.

CASE 2.—Henry B., æt. 20 (Vol. 121, No. 304), was in Guy's Hospital in 1891. He gave no important family history. He had never received a head injury. He was a broker's clerk, and had led a very dissipated life. He had had gonorrhœa five times, and had drunk spirits freely. For five years previous to admission he had suffered from hydruria, getting up five or six times each night to micturate. Lately he had been getting weaker; his appetite was poor; he was constipated, and extremely thirsty. He was treated at different times with valerianate of zinc; with opium; and with infusion of valerian; but nothing made him better. He passed from 150 to 278 ounces of urine daily, the specific gravity being 1001 to 1007. The reaction was acid; it contained no albumen, nor sugar; and the urea ranged from 250 to 650 grains per diem.

CASE 3.—Joseph C., æt. 3 (Vol. 123, No. 363), was in Guy's Hospital in 1891. His maternal grandmother had "diabetes"; as also had cousins on his mother's side. Since he was six months

old, the patient had suffered from hydruria; and he used to drink anything that came in his way. Thus it came about that he was admitted for drinking vitriol out of a bottle. In hospital, from two to six pints of urine were collected a day, averaging about four pints. The specific gravity was between 1006 and 1008. It was acid in reaction; and never contained sugar, nor albumen.

CASE 4.—Henry W., æt. 48 (Vol. 120, No. 264, and Vol. 125, No. 278), was in Guy's Hospital both in 1891 and in 1892. The history does not say how long he had been affected. There was no important family history. He had been a very heavy beer drinker; there was no history of head injury. He suffered from intense thirst and excess of urine, but was admitted upon the first occasion for abdominal pain and vomiting, and upon the second for profuse diarrhoea. His cardiac and respiratory systems were normal, but his urine amounted to between 200 and 280 ounces per diem, of a specific gravity varying from 1002 to 1010. It was acid, contained no albumen, casts, nor sugar, and the urea was very variable, between 500 and 1029 grains per diem. His temperature was normal throughout. His pulse was not of high tension. He was treated with codeine, but his thirst and hydruria were not relieved.

CASE 5.—John S., æt. 6 (Vol. 138, No. 27), was in Guy's Hospital from October 13th, 1893, to April 27th, 1894. The family history is unimportant. He began to suffer from thirst and excess of urine three months before admission, without evident cause. An average of 100 ounces urine were collected from him daily, of acid reaction, and specific gravity 1005 to 1015. No albumen was present. The case was regarded as one of diabetes insipidus, because the specific gravity was low. *But sugar was almost always present* in the urine, and gave the phenyl hydrazine test. Upon restricted diet he frequently passed little sugar, the amount per diem varying from none at all to 400 grains. Upon full diet he passed from 700 to 1500 grains of sugar daily. Treatment by valerian was adopted with no effect. The patient did better on full diet in spite of the increased quantity of sugar in his urine.

This appears an anomalous case. It differs from all the others in the presence of sugar. It may have been an instance of diabetes mellitus with a low specific gravity urine, but it may be a rarer condition of diabetes insipidus, accompanied by glycosuria.

CASE 6.—Edward W., æt. 23 (Vol. 136, No. 403), was in Guy's Hospital from November 29th to December 10th, 1894. He gave no important family history. He was a clerk in a brewery, but was very abstemious. He did not drink alcohol. He worried a great deal. In the previous July he had suffered from headache, sleeplessness and weariness. He gave a three months' history of thirst and excess of urine. He was a very nervous man. He passed from 200 to 300 ounces of urine daily, sometimes acid, sometimes alkaline. The specific gravity was constantly 1004. The urea varied from 300 to 900 grains per diem. No albumen, nor sugar, was ever found. He was treated with Mist. Acidi Co. (G.H.P.), but showed no improvement.

CASE 7.—William W., æt. 36 (Vol. 140, No. 156), was in Guy's Hospital from May 6th to May 19th 1895. His father suffered from gout, but there is no other family history. He was a carpenter by trade; and in January, 1892, had fallen off a scaffold, and suffered concussion. It was not till March, 1894, that he noticed his thirst and excess of urine, which came on after a "shivering fit." A note, added to the history, says that he used to drink five gallons a day before this rigor (? as a result of the concussion). In the hospital, he passed from 150 to 250 ounces of urine per diem, of a specific gravity 1002 to 1006; it was neutral, contained from 200 to 250 grains of urea, and never any casts, albumen, or sugar. He was given valerian, but with no improvement.

CASE 8.—Winifred B., æt. 26 (Vol. 147, No. 367), was in Guy's Hospital from July 10th to September 19th, 1896. The family history does not bear upon the condition. The patient was not in the least neurotic, and could assign no cause for the thirst and urine excess. She was a servant, and gave a three months' history of her symptoms. In hospital, she drank from eleven to twenty-five pints a day, and from 175 to 360 ounces of urine were

collected. The specific gravity was always about 1003; the reaction acid; and the urea varied from 300 to 370 grains; no sugar nor albumen was present. After trying suprarenal tabloids, which made her worse, and valerianate of zinc, which had no effect, she was treated by Faradism, one pole being placed over the lumbar spine, and the other over the site of each kidney alternately. She improved, and felt a great deal better for the electric application; though her urine decreased but slightly in amount.

CASE 9.—Louisa W., æt. 50 (Vol. 151, No. 206), was in Guy's Hospital from July 14th to September 2nd, 1897. Her father died of epilepsy. In May, 1897, she was frightened by a drunken man. Since then she had had hydruria and thirst. She had neither before. The urine varied above and below 200 ounces daily, of a specific gravity 1000 to 1002; acid reaction; urea 250 to 500 grains, and no albumen nor sugar. Treatment by valerianate of zinc; by asafoetida; by opium; and by atropine was equally unsuccessful.

CASE 10.—Ellen K., æt. 39 (Vol. 161, No. 162; and Vol. 167, No. 399), was in Guy's Hospital from April 28th to May 14th, 1899, and from December 6th to December 19th, 1900. Upon each occasion she came in for "asthma" and emphysema. Her mother died of "asthma." The patient had had "thirsty diabetes" since she was eight years old. She said it was brought on by a fright. Her urine amounted to 150 to 340 ounces per diem, with a specific gravity 1000 to 1004; acid reaction; no sugar, nor albumen; and very variable urea, 75 to 500 grains daily. She had grown used to her "thirsty diabetes," and was only troubled by her cough.

CASE 11.—Minnie H., æt. 11 (Vol. 166, No. 187), was in Guy's Hospital from May 24th to August 6th, 1900. Her maternal grandmother died of "diabetes." The patient gave no cause for her thirst and hydruria. The symptoms began three months before admission. She had a poor appetite. Her urine varied between 100 and 275 ounces daily; of a specific gravity 1002 to 1005; acid reaction; urea, 250 to 450 grains; and no albumen nor

sugar. She was treated with suprarenal extract, and with ergot, but without improvement.

Of the eleven cases, seven were men, four were women. The average age was twenty-five, the youngest being three and the oldest fifty. Few gave an important family history. The symptoms, and the condition of the urine, were very similar in all. In one case only, case 5, was sugar found. Beale (3) says: "There is generally no sugar; but I have met with a few instances in which urine of a very low specific gravity (1004) contained an appreciable amount of sugar." Case 10 exemplifies the possible prognosis; the patient had had diabetes insipidus for thirty years. In one instance only, case 7, is there any history of a head injury; and even this patient did not connect his symptoms with his concussion.

*Cases of diabetes insipidus associated with injury or lesion of the medulla oblongata.*

Head injuries are common, hydruria as a consequence is rare; few instances have been published. The following are cases of tumours or injuries of the medulla oblongata, associated with thirst and excess of urine.

CASE A.—A man, æt. 33, was under the care of Dr. Alex. Robertson (14) with definite symptoms of a tumour in the region of the fourth ventricle. The history extended over sixteen months. He worked on board a boat. He came under observation passing 200 to 260 ozs. urine a day, specific gravity 1002 to 1003, acid reaction, without sugar or albumen. Under treatment by the continuous current, applied to the medullary region, his urine fell to 78 or 88 ozs. per diem; specific gravity 1010 to 1012. He went back to work. Later he had a relapse, but, being again treated by the continuous current, he improved once more, and returned to work.

CASE B.—A woman mentioned by Dr. Ralfe (13) as suffering from hydruria, without albumen or sugar. One day she died suddenly, with cerebral symptoms, and a small aneurysm was found ruptured at the base of the brain.

CASE C.—Mr. Battle (2) mentions a patient who was brought in o St. Thomas's Hospital unconscious, and who passed many pints of urine a day. There is no note of the specific gravity, nor of the absence of sugar. Post-mortem the lesion found was a gumma of the floor of the fourth ventricle.

CASE D.—A man, æt. 35, was studied by Dr. Strubell (22) in Jena. This man fell backwards and struck his right ear against a piece of wood. He did not lose consciousness but felt stunned, and within half an hour hydruria and thirst came on. He passed many litres of urine, of low specific gravity, and without sugar. At Jena the condition was at first regarded as primary thirst and secondary hydruria, but Strubell showed the reverse to be the case by examining the specific gravity of the blood; the results, which are as follows, are similar to those obtained in the present case, but the change in specific gravity, when fluid by the mouth was restricted, was even more marked :—

	Specific gravity of blood.	Red Corpuscles.	White Corpuscles.	Hæmoglobin. Per cent.
Water freely taken	1057	5,500,000	11,000	110
When water restricted	1077	5,950,000	11,500	110

Strubell partly investigated the nitrogen metabolism in his case, for ten days only. He determined neither the urea nor the uric acid, but only the total nitrogen. As in the present instance, he found the nitrogen excretion to be normal; but he concluded from his figures that the nitrogen elimination was greatly reduced when fluid was severely restricted. His patient received 14·5 grams nitrogen in his food each day; and, when fluid was freely given, 13 grams nitrogen were recovered in the urine, and 1·2 grams in the fæces daily. But when the drinking water was strictly limited, the nitrogen in the urine fell to 8·4 grams, though the food was the same as before. He concluded that restriction of water caused restriction of nitrogen metabolism. In the present instance no such change has been observed. On the last two days of the research, when fluid was greatly restricted, more nitrogen was recovered than was given in the food; and the proportions of urea and of uric acid to

the total nitrogen in the urine were unaltered (see Table I. and Table IV.).

Strubell further showed that no excess of sodium chloride was passed by his patient.

Three further instances of diabetes insipidus following injury are recorded by Senator (17). This writer has recorded seventy-six cases of the disease, but in three only (= 4 per cent.) did it follow fall or injury to the head. He quotes Cantani as saying that diabetes mellitus, on the other hand, is preceded in 10 per cent. of instances by a history of injury. Senator himself collected 1090 cases of diabetes mellitus in his paper, of whom eleven only, or 1 per cent., gave a direct history of injury. He concludes that head injury is quite a rare cause of either form of diabetes; may cause either; but causes the insipid form somewhat more often than the sweet.

Full references to the literature upon the subject previous to 1879 will be found in the article by Senator in Ziemmsen's *Handbuch* (18), and for the most recent literature the papers by Strubell (22) and by Senator (17) should be consulted.

#### THE PATHOLOGY OF DIABETES INSIPIDUS.

Dr. Saundby (15) has recorded three instances in which patients suffering from diabetes insipidus have died with symptoms of uræmia, and at the autopsies the kidneys were found sacculated and wasted, with adherent capsules, and microscopic changes like those of chronic interstitial nephritis. But these changes he regarded as mechanical, and due to the constant accumulation of excess of urine in the bladder, with consequent backward pressure upon the kidneys.

The rapid onset of hydruria and thirst after a head injury, as in the present instance and in case D, indicates that the symptoms depend on no structural alteration in the kidney cells.

Claude Bernard proved by experiment on animals that the medulla oblongata contains two centres, injury to one of which produces glycosuria, injury to the other, hydruria. The latter is situated a little higher than the former; both are near the vaso-motor centre. It is often assumed that disturbance of the second of the two is the cause of diabetes insipidus.



Just as Claude Bernard experimentally disturbed this centre, so head injuries may do so. It is, perhaps, surprising that head injuries do not do so more constantly, and it is perhaps still more surprising that head injury of a gross kind can disturb the hydruria centre, leaving the adjacent glycosuria point unharmed.

The sudden driving of cerebrospinal fluid down the iter into the fourth ventricle is possibly the immediate cause of the disturbance in such a case as Strubell's (22), but more direct injury to the medulla seems likely to result from a fractured base; or from a tumour, aneurysm, or gumma, as in cases A, B and C.

But, granting that a centre in the medulla has been interfered with, this does not fully explain the hydruria. The older theory supposed the polydipsia to be primary, excess of watery urine to be the consequence. This is no longer tenable, in view of the great rise in the specific gravity of the blood when drinking is restricted. The hydruria is primary, the polydipsia results. For some reason the kidney is no longer able to restrict the passage of water. Theory suggests that this depends upon vasomotor disturbance. In cases of granular kidney it is believed that increase in the general blood-pressure is the cause of the hydruria. In the present patient, however, observations upon his radial blood-pressure showed it to be neither higher nor lower than that of healthy students. It would seem, therefore, that, if any vasomotor nerves are at fault, they must be those of the kidney itself. It is difficult to conceive how a gross lesion of the brain, whether injury or tumour, can pick out just that portion of the vasomotor centre which controls the renal vessels, leaving all the rest unaltered.

Lazarus Barlow (9) mentions another possibility, namely, that the hydruria may arise from defective re-absorption rather than from excessive secretion on the part of the kidney. The two chief views as to the normal kidney action are shortly as follows. The first, that of Heidenhain, is widely accepted, and holds that the epithelium of the renal tubules actively secretes the organic constituents of the urine, adding them to the water and salts which, by modified filtration, pass through

the glomeruli. Sobieranski (20) upon repeating Heidenhain's sulphindigotate of soda experiment, draws an opposite conclusion, namely, that all the constituents of the urine, water, salts, and urea, come through the glomeruli in very dilute solution; and that it is the function of the tubal epithelium to actively reabsorb a large part of the water, and return it to the blood. That in either case the renal epithelium must play an active part was proved by Dreser (6), who calculated the amount of positive work which must be done in separating the urine from the blood.

Sobieranski's theory would explain the pathology of diabetes insipidus, not as an excessive transudation through the glomeruli due to local vasodilatation, but as a defective power of reabsorbing water on the part of the tubal epithelium; so that urine reached the pelvis of the kidney as dilute as it left the glomeruli. In granular kidney, as Lazarus Barlow (9) points out, a similar hydruria might be expected, because here the renal epithelium has partly atrophied. Physiology has not demonstrated a centre in the medulla directly controlling kidney cell activity; but it is no more difficult to conceive that a head injury may interfere, through the nervous system, with the normal functions of the renal cells, than it is to understand how such an injury can affect that part of the vasomotor centre only which controls the renal vessels.

Both views are hypothetical; but, upon the theory of excessive excretion, one might expect those inorganic constituents of the urine which transude with the water to be increased: for example, the sodium chloride. But Strubell showed that, in his case, a normal quantity of salt passed with the urine. The other constituents are normal in the present instance; the quantities of urea, of uric acid, and of nitrogen, are those of health, both in total amount and in relative proportions. The water alone was at fault; a condition which would follow from Sobieranski's theory as to healthy kidney action, if the urine were excreted by the glomeruli in a normal manner, but failed to become concentrated by the tubal epithelium.

#### TREATMENT.

During the research, no experiments were carried out from the point of view of treatment. That harm need not follow a full diet has been shown by the normal metabolism of both proteid and carbohydrate in the present instance.

In the few cases which have occurred in Guy's Hospital, no medicinal treatment has been effective. Valerian and asafoetida were administered in most cases, with no result. Ergot (case 11), codeine (case 4), opium (case 2), atropine (case 9), suprarenal extract (cases 8 and 11), were equally unsuccessful. Ergot and suprarenal extract even aggravated the condition; that they should not produce benefit might be expected if the pathology of the diabetes is not of vasomotor origin, but is dependent on defective resorptive power in the renal epithelium.

In two cases only was amelioration seen, and in each of these (case 8 and case A.) electrical treatment was adopted; in case 8, Faradization to the region of the kidneys; in case A., the continuous current over the position of the medulla oblongata.

Strubell (22) remarks that, after profuse perspiration, his patient lost both thirst and hydruria for a time. The relation between the skin and the kidneys is well known; that perspiration should diminish the amount of urine, even in diabetes insipidus, seems not unlikely, but it is almost a paradox that it should also diminish the thirst. When the skin is acting freely, perhaps the renal epithelium recovers its power of reabsorbing water and returning it to the blood. This suggests that, in treating the condition, diaphoretics such as pilocarpine or Turkish baths may be useful; hitherto no instance of this treatment has been recorded, but in future cases it would be of great interest to try it.

#### GENERAL CONCLUSIONS.

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These are, that, in the patient investigated—

1. Diabetes insipidus was immediately associated with a fracture to the base of his skull.
2. His condition was that of simple hydruria.

3. His proteid metabolism was normal.
4. His power of assimilating all kinds of carbohydrate was good; glycosuria was not produced by administering more than 700 grams of dry carbohydrate in twenty-four hours; he assimilated both starch, and cane sugar, and glucose.
5. His urea constituted 88.5 per cent. of all the nitrogen in his urine.
6. Upon milk diet, his uric acid was very low; upon fuller diet it was high.
7. The uric acid was readily estimated by the Hopkins' permanganate process, though the urine was very dilute.
8. Of all the fluid he took by the mouth, 90 per cent. was recovered in the urine.
9. His hydruria was primary, his polydipsia secondary to it; because, when fluid was restricted to three litres a day, the specific gravity of the blood rose from 1061 to 1067; and both red corpuscles and hæmoglobin showed corresponding concentrations.
10. That, in its relation to glycosuria, traumatic diabetes insipidus does not appear to differ from non-traumatic.
11. That instances of head injury followed by hydruria are far from common.
12. That full diet is, in some cases at least, permissible; that medicinal treatment is less effective than electrical; and that possibly Turkish baths or diaphoretics would give relief.

In conclusion, the writers give their best thanks to Dr. Hale White, for permission to investigate the case; to Dr. Perry, for the use of Stephen ward private room; to the Matron of Guy's Hospital, Sister Stephen, and her Nurses for their hearty co-operation; and to Dr. Pembrey for giving every possible facility for the analytical work in Guy's Hospital Physiological Laboratory. The expenses of the research were defrayed out of a grant from the Scientific Grants Committee of the British Medical Association to Dr. Pembrey.

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Reference may also be made to a paper by T. B. Fitcher, M.B. (Tor.), entitled, "Diabetes Insipidus, with a report of five cases," which has appeared in the *Johns Hopkins Hospital Reports* for 1902. It contains some metabolism investigations, but unfortunately the present paper was already in the press before Mr. Fitcher's work was published.

TABLE I.

	THE URINE.			THE FLUID IN THE FOOD, IN C.C.					
	Amount in c.c.	Reaction to litmus.	Specific gravity by bottle.	As Milk.	As Water.	As Egg.	As Drugs.	Total Fluid in Food.	% of Fluid consumed retained in urine
During 24 hours ending at 10 a.m. on:—									
Sept. 12	8565	Amphoteric	1008.9	4000	5143	—	142	9285	92.24 %
13	8840	Acid	1003.9	3762	5115	—	142	9019	92.47 "
14	7885	"	1003.7	3996	5683	—	142	9821	80.29 "
15	8340	"	1004.0	3996	4547	—	142	8685	96.03 "
16	8190	"	1003.3	4416	5115	—	142	9673	84.67 "
17	6870	"	1003.4	4331	4504	—	142	8977	76.53 "
18	8215	Alkaline	1003.3	2986	5115	—	85	8186	100.36 "
19	7895	"	1004.0	2301	6536	50	85	8872	88.99 "
20	6987	Amphoteric	1003.4	2193	5683	100	85	8061	85.56 "
21	8230	"	1003.9	2244	7388	100	85	9817	83.83 "
22	8479	Acid	1004.2	2143	6252	100	85	8580	98.82 "
23	7915	Alkaline	1004.1	2232	6252	75	85	8544	91.57 "
24	8557	"	1003.5	2153	6820	150	85	9208	92.93 "
25	8140	Amphoteric	1004.8	2116	6820	125	85	9146	89.00 "
26	8760	"	1004.3	2177	6820	100	85	9182	95.40 "
27	7533	"	1003.7	1998	6252	150	85	8485	98.78 "
28	6748	Alkaline	1004.1	2148	6252	150	85	8635	78.15 "
29	7805	Amphoteric	1002.8	1069	6820	75	85	8049	96.97 "
30	6350	Alkaline	1004.0	1598	5683	150	85	7516	84.48 "

TABLE I.—continued.

THE URINE.			THE FLUID IN THE FOOD, IN C.C.						o/o of Fluid consumed re- covered in urine
Amount in c.c.	Reaction to litmus:—	Specific gravity by bottle.	As Milk.	As Water.	As Egg.	As Drugs.	Total Fluid in Food		
During 24 hours ending at 10 a.m. on:—									
Oct. 1	1705	Amphoteric	1003.3	2174	5115	150	85	7524	102.40 %
2	7780	Alkaline	1008.0	2248	6252	100	85	8685	89.00 "
3	8000	"	1004.1	1726	6252	150	85	8218	97.41 "
4	8510	"	1004.5	1778	6820	100	85	8783	96.89 "
5	8117	Amphoteric	1004.4	2172	5683	125	85	8065	100.65 "
6	6780	Alkaline	1004.6	2120	5683	100	85	7988	84.25 "
7	6310	"	1005.3	1825	5115	150	85	7175	87.94 "
8	6995	"	1005.1	2226	5399	100	85	7810	89.57 "
9	7162	"	1004.0	2125	5683	100	85	7993	89.60 "
10	7840	Amphoteric	1004.0	2252	5683	75	85	8095	96.85 "
11	5687	Alkaline	1005.1	1450	5115	75	85	6725	84.56 "
12	7370	"	1004.7	2224	5683	100	85	8092	91.08 "
13	6470	Amphoteric	1004.4	2210	5115	75	85	7485	86.44 "
14	3544	"	1007.5	1067	1705	125	85	2972	119.24 "
15	3270	Acid	1007.5	1107	1705	150	85	3047	107.32 "
Average	7390 c.c.			2366 c.c.	5583 c.c.			8132 c.c.	90.87 %

TABLE II.

## THE NITROGEN EXCRETED IN URINE AND FÆCES

(IN GRAMMES).

During 24 hours ending at 10 a.m.	Nitrogen as Urea.	Nitrogen as Uric Acid.	Nitrogen as "other bodies."	Total Nitrogen in Urine.	Total Nitrogen in Fæces.	Total Nitrogen in Urine and Fæces.
Sept. 12	14·0824*	0·0739	1·4223	15·5286	0·9193	16·4479
13	14·9037	0·0433	2·8808	17·8278	2·6533	20·4811
14	13·9130	0·0481	2·3929	16·3540*	1·4054	17·7594
15	15·8737*	0·0862†	1·5266	17·4865	3·3233	20·8098
16	16·2600*	0·0454	0·7451	17·0505*	2·9002	19·9507
17	13·0267	0·0648	1·7500	14·8415	1·9001	16·7416
18	15·5772	0·0628	0·7764	16·4164	2·7191	19·1355
19	16·2779	0·2175	0·6458	17·1412	3·5705	20·7117
20	13·7031	0·2611	2·1396	16·1038	1·5357	17·6395
21	16·3156	0·3231	1·3751	18·0138	3·1675	21·1813
22	16·3407	0·5500	1·3056	18·1963	3·7647	21·9610
23	16·4528*	0·3911	0·4775	17·3214	2·1738	19·4952
24	17·6636	0·4828	0·4768	18·6232	3·0063	21·6295
25	17·9793	0·4958	1·4624	19·9375	3·2389	23·1764
26	18·0509	0·3721	0·7010	19·1240	2·8658	21·9898
27	15·0830*	0·4867	1·5482	17·1179	2·7712	19·8891
28	13·6660†	0·4476	0·2017	14·3153	2·3447	16·6600
29	11·3423	0·4211	1·2446	13·0080	2·3868	15·3948
30	10·9627	0·4347	1·5849	12·9823	1·9636	14·9459
Oct. 1	13·1377	0·3410	1·5484	15·0271	1·6799	16·7070
2	14·3588	0·3387	1·1008	15·7983	1·6268	17·4251
3	12·9008*	0·3215	2·9812	16·2035	2·6515	18·8550
4	14·0383	0·1681	1·9040	16·1104	1·1390	17·2494
5	12·9281	0·2396	2·9692	16·1369	2·7888	18·9257
6	12·6524	0·3309	1·9638	14·9471	1·8748	16·8219
7	14·1780*	0·3559	1·5598	16·0937	2·0979	18·1916
8	14·9624*	0·3761	2·2434	17·5819	2·2006	19·7825
9	11·8852	0·3051†	1·9087	14·0990	1·9585	16·0575
10	13·5009	0·3365	2·2411	16·0785	1·9249	18·0034
11	12·1397	0·2897	1·4821	13·9115	1·3382	15·2497
12	12·3253†	0·3388	2·0991	14·7632	2·4217	17·1849
13	12·6704	0·2255	1·1775	14·0834	5·2647	19·3481
14	11·6271	0·2562	1·2735	13·1568	1·0281	14·1849
15	12·5800	0·1860	1·7975	14·5635	1·1467†	15·7102
Average	14·2090	0·2861	1·5561	16·0812	2·3456	18·3968

\* = Highest estimation taken.

† = One estimation only.



TABLE III.

For 24 hours, ending at 10 a.m. on:--	FÆCES.		IF THE TOTAL NITROGEN IN URINE = 100, THEN:—			URIC ACID.	
	Number of times bowels were open.	Weight of Dried Fæces.	Urea Nitrogen =	Uric Acid Nitrogen =	Other bodies Nitrogen =	Ratio of Uric Acid as such to Urea as such.	Ratio of Uric Acid Nitrogen to Total Nitrogen in Urine.
Sept. 12	2	40.25 grms.	90.364 %	0.476 %	9.160 %	1 to 136	1 to 210
13	3	109.70 "	83.600 "	0.242 "	16.158 "	1 " 246	1 " 412
14	3	32.90 "	85.074 "	0.294 "	14.632 "	1 " 207	1 " 340
15	4	67.05 "	90.778 "	0.493 "	8.729 "	1 " 132	1 " 203
16	2	53.50 "	95.365 "	0.266 "	4.369 "	1 " 256	1 " 376
17	3	42.20 "	87.773 "	0.436 "	11.791 "	1 " 144	1 " 229
18	3	54.50 "	94.890 "	0.382 "	4.728 "	1 " 177	1 " 261
19	5	55.00 grms.	94.966 %	1.268 %	3.766 %	1 to 57	1 to 79
20	2	29.40 "	85.092 "	1.621 "	13.287 "	1 " 37	1st day of Liebig 1 to 62
21	2	51.20 "	90.574 "	1.793 "	7.633 "	1 " 36	1 " 56
22	2	54.05 "	89.802 "	3.023 "	7.175 "	1 " 21	1 " 33
23	1	38.25 "	94.986 "	2.358 "	2.756 "	1 " 30	1 " 44
24	3	45.20 "	94.848 "	2.592 "	2.560 "	1 " 26	1 " 39
25	3	47.10 "	90.180 "	2.486 "	7.334 "	1 " 26	1 " 40
26	2	51.30 "	94.388 "	1.946 "	3.666 "	1 " 35	1 " 51
27	2	40.55 "	88.112 "	2.843 "	9.045 "	1 " 22	1 " 35
28	1	40.90 "	95.465 "	3.126 "	1.409 "	1 " 22	1 " 32
29	3	41.15 "	87.196 "	3.237 "	9.567 "	1 " 19	1 " 31
30	3	28.55 "	84.445 "	3.348 "	12.207 "	1 " 18	1 " 30
Oct. 1	2	26.10 "	87.426 "	2.269 "	10.305 "	1 " 28	1 " 35
2	2	27.00 "	90.886 "	2.144 "	6.970 "	1 " 30	1 " 47
3	2	42.05 "	79.617 "	1.984 "	18.399 "	1 " 29	1 " 50
4	1	17.50 "	87.138 "	1.043 "	11.819 "	1 " 60	1 " 96
5	2	45.05 "	80.115 "	1.485 "	18.400 "	1 " 39	1 " 67
6	2	31.90 "	84.648 "	2.214 "	13.138 "	1 " 27	1 " 45
7	2	32.95 "	88.096 "	2.211 "	9.693 "	1 " 28	1 " 45
8	2	35.70 "	85.100 "	2.139 "	12.761 "	1 " 28	1 " 47
9	2	33.85 "	84.298 "	2.164 "	13.538 "	1 " 28	1 " 46
10	2	27.90 "	83.966 "	2.093 "	13.941 "	1 " 29	1 " 48
11	3	20.35 "	87.262 "	2.083 "	10.655 "	1 " 30	1 " 48
12	3	40.55 "	83.485 "	2.295 "	14.220 "	1 " 26	1 " 44
13	3	91.70 "	89.968 "	1.672 "	8.360 "	1 " 32	1 " 75
14	2	16.15 "	88.374 "	1.947 "	9.679 "	1 " 32	1 " 51
15	1	16.45 "	86.882 "	1.277 "	12.341 "	1 " 48	1 " 78
Average, when diet milk only			...	...	...	1 to 174	1 to 272
Average, when more than milk in diet			...	...	...	1 to 29	1 to 46
Average for the whole period...			88.53 %	1.78 %	9.69 %	1 to 35	1 to 56

Period of purely milk diet

TABLE IV.

For the 24 hours ending at 10 a.m. on :—	The Nitrogen contained in the food daily, in grammes :—						The Nitrogen recovered in Urine and Fæces.	The Nitrogen Balance.		Patient's weight in Grammes.	
	In Milk.	In Biscuit.	In Butter.	In Liebig.	In Eggs.	In Arrow- root.		Total.	Difference between in-take and out-put of Nitrogen daily.		Progressive deficit of Nitrogen.
Sept. 12	21.4253	—	—	—	—	—	21.4253	16.4479	+ 4.9774	4.9774	69.970
13	19.9808	—	—	—	—	—	19.9808	20.4811	— 0.5003	4.4771	69.514
14	20.8795	—	—	—	—	—	20.8795	17.7594	+ 3.1201	7.5972	70.650
15	20.8795	—	—	—	—	—	20.8795	20.8098	+ 0.0697	7.6669	70.691
16	23.0735	—	—	—	—	—	23.0735	19.9507	+ 3.1238	10.7897	71.554
17	22.6900	—	—	—	—	—	22.6900	16.7416	+ 5.8884	16.6781	72.010
18	15.6020	—	—	—	—	—	15.6020	19.1355	— 3.5335	13.1446	71.500
19	11.5002	1.8467	0.0859	—	1.9970	—	15.3798	20.7117	— 5.3319	7.8127	71.214
20	11.4585	1.7070	0.0859	3.0096	3.9890	—	20.2490	17.6395	+ 2.6095	10.4222	71.840
21	11.7250	2.1690	0.1089	5.9894	8.9880	—	23.9743	21.1813	+ 2.7930	13.2152	73.090
22	11.1972	2.0040	0.1278	5.5887	3.9880	—	22.9000	21.9610	+ 0.9397	14.1549	71.101
23	11.6622	0.7137	0.1233	4.7598	2.9910	—	20.2500	19.4952	+ 0.7548	14.9097	72.575
24	11.2492	1.2702	0.1518	6.6766	5.9820	—	25.3298	21.6295	+ 3.7003	18.6100	72.234
25	11.0565	1.2811	0.1416	5.4061	4.9850	—	23.8703	23.1764	— 0.8061	18.8039	72.234
26	11.3750	1.2735	0.1616	5.2277	3.9880	—	22.0258	21.9698	+ 0.0360	18.3399	72.291
27	11.5605	1.4509	0.2160	4.9093	5.9820	—	24.1187	19.8891	+ 4.2296	22.5695	73.812
28	12.4283	1.1079	0.1897	9.3169	5.9820	—	23.0248	16.6600	+ 6.3648	28.9343	74.502
29	6.1853	1.1413	0.2141	7.2060	2.9910	—	17.7377	15.3948	+ 2.3429	31.2772	73.992
30	9.2462	1.1363	0.2174	2.4503	5.9820	—	19.0322	14.9459	+ 4.0863	35.3635	74.388

TABLE IV. — continued.

For the 24 hours ending at 10 a.m. on:—	The Nitrogen consumed in the food daily, in grammes :—							The Nitrogen recovered in Urine and Faeces.	The Nitrogen Balance.		Patient's weight in Grammes.
	In Milk.	In Biscuit.	In Butter	In Liebig.	In Eggs.	In Arrow- root.	Total.		+ = deficit in urine & faeces. — = surplus in urine & faeces.		
									Difference between intake and out-put of Nitrogen.	Progressive deficit of Nitrogen.	
Oct. 1	12.5788	1.1363	0.1704	2.1317	5.9820	—	21.9892	16.7070	+ 5.2822	40.6457	73.992
2	13.0073	1.1238	0.2327	4.7428	3.9880	—	23.0946	17.4251	+ 5.6695	46.3152	74.620
3	9.9867	1.2945	0.3419	2.3906	5.9820	—	19.9957	18.8550	+ 1.1407	47.4559	74.900
4	10.2876	0.9790	0.1783	4.5762	3.9880	—	20.0091	17.2494	+ 2.7597	50.2156	74.620
5	12.5672	0.8418	0.2055	2.7756	4.9850	—	21.3751	18.9257	+ 2.4494	52.6650	74.050
6	12.2667	1.1490	0.2490	3.3214	3.9880	—	20.9681	16.8219	+ 4.1462	56.8112	74.678
7	10.5595	1.0827	0.2109	5.0893	5.9820	—	22.9244	18.1916	+ 4.7328	61.5440	74.843
8	12.8795	1.3087	0.2688	2.9902	3.9880	0.0379	21.4731	19.7825	+ 1.6906	63.2346	74.900
9	12.2955	0.8535	0.0741	2.6404	3.9880	—	19.8515	16.0375	+ 3.7940	67.0386	75.190
10	13.0803	0.8719	—	6.1203	2.9910	0.1419	23.1554	18.0084	+ 5.1520	72.1806	75.900
11	8.3898	0.5706	—	5.0847	2.9910	0.1173	17.1534	15.2497	+ 1.9037	74.0843	75.410
12	12.8680	0.5606	—	2.3558	3.9880	0.1285	19.9009	17.1849	+ 2.7160	76.8003	75.188
13	12.7870	0.5757	—	3.1208	2.9910	0.1665	19.6410	19.3481	+ 0.2929	77.0932	75.900
14	6.1157	1.1530	—	2.7989	4.9850	0.0523	15.1049	14.1849	+ 0.9200	78.0132	74.388
15	6.4051	1.7254	—	—	5.9820	—	14.1125	15.7102	— 1.5977	76.4155	74.050
Average							20.6505	18.4030	+ 2.2475		

TABLE V.

During the 24 hours ending at 10 a.m. on:—	FOOD CONSUMED DAILY.							
	Milk, in c.c.	Breakfast Biscuit, in grammes.	Butter, in grammes.	Liebig, in grammes.	Cane Sugar, in grammes.	Number of Eggs.	Grape Sugar, in grammes.	Arrow- root, in grammes.
Sept. 12	4000	—	—	—	—	—	—	—
13	3762	—	—	—	—	—	—	—
14	3996	—	—	—	—	—	—	—
15	3996	—	—	—	—	—	—	—
16	4416	—	—	—	—	—	—	—
17	4331	—	—	—	—	—	—	—
18	2986	—	—	—	—	—	—	—
19	2201	110·35	10·25	—	—	2	—	—
20	2193	102·00	24·50	35·25	—	4	—	—
21	2244	129·25	31·05	70·15	—	4	—	—
22	2143	119·75	36·45	65·40	—	4	—	—
23	2232	42·65	35·15	55·75	—	3	—	—
24	2153	75·90	43·28	78·20	—	6	—	—
25	2116	76·55	40·37	63·32	—	5	—	—
26	2177	76·10	46·10	61·23	—	4	—	—
27	1998	86·70	61·60	57·50	578·05	6	—	—
28	2148	66·20	54·10	38·85	401·25	6	—	—
29	1069	68·20	61·05	84·40	225·40	3	—	—
30	1598	67·90	62·00	28·70	296·80	6	—	—
Oct. 1	2174	67·90	34·50	24·85	347·95	6	—	—
2	2248	67·15	47·10	55·55	173·25	4	—	—
3	1726	77·35	69·20	28·00	102·90	6	266·30	—
4	1778	58·50	36·10	53·60	—	4	193·50	—
5	2172	50·30	41·60	29·75	274·40	5	—	—
6	2120	68·30	50·40	35·60	265·45	4	95·45	—
7	1825	64·70	42·70	54·55	130·55	6	—	—
8	2226	78·20	54·70	32·05	239·30	4	46·80	32·30
9	2125	51·00	15·00	28·30	40·25	4	205·80	—
10	2252	52·10	—	65·60	66·75	3	209·10	120·90
11	1450	34·10	—	54·50	31·90	3	100·10	99·95
12	2224	33·50	—	25·25	40·60	4	154·10	109·50
13	2210	34·40	—	33·45	72·10	3	127·00	141·90
14	1057	68·90	—	30·00	—	5	—	44·60
15	1107	103·10	—	—	—	6	—	—

**COMPOSITION OF FOOD STUFFS USED:—**

*Milk:—*

On September 11th, and part of September 12th, ordinary cow's milk was used. Three different supplies were used; each was analysed. The nitrogen content was—

- |    |                      |     |                  |
|----|----------------------|-----|------------------|
| 1. | In the first supply  | ... | 0·5376 per cent. |
| 2. | In the second supply | ... | 0·5387 per cent. |
| 3. | In the third supply  | ... | 0·5387 per cent. |

From September 12th to September 27th, milk sterilized, and all from the same churn, was used. The composition was as follows :—

Nitrogen ... 0.5225 per cent. (by direct Kjeldahl).  
Carbohydrate 4.66 grams per 100 c.c. } by Aylesbury Dairy  
Fat ... 4.92 grams per 100 c.c. } Co. Analyst.

From September 27th to October 15th, a similar, but fresh, supply of milk was used, the composition being:—

Nitrogen	...	0.5786 per cent. (by direct Kjeldahl).	
Carbohydrate	4.75 grams per 100 c.c.		} certified by Aylesbury Dairy Co. Analyst.
Fat	...	3.69 grams per 100 c.c.	

**Biscuits :—**

These were called "breakfast biscuits," crisp, and not sweet. They contained:—

Nitrogen ... 1.6735 per cent. (by direct Kjeldahl).  
Carbohydrate 75 per cent. (Stevenson & Murphy, Ref. 5).

*Butter :—*

This contained :—

Nitrogen ... 1st lot, 0.3507 per cent. (by direct Kjeldahl).  
2nd lot, 0.4941 per cent. ..

Fat ... 83·5 per cent. (Diet Tables, Ref. 5).

*Liebig :—*

This contained, by direct Kjeldahl, 8.5378 per cent. nitrogen. The same tin was used from September 19th to October 4th. A second tin was used for the rest of the time, containing, by Kjeldahl, 9.3296 per cent. nitrogen.

*Eggs :—*

Average weight, with shell, unboiled, 54·865 grams ; this was the average of 129 eggs.

An average egg contained :—

Nitrogen ... 0·9970 gram (by direct Kjeldahl).

Fat ... 6·03 grams (Stevenson & Murphy, Ref. 5).

Water ... 25 c.c.

*Cane sugar :—*

Contained 100 per cent. carbohydrate.

*Grape sugar :—*

Contained 100 per cent. carbohydrate.

*Arrowroot :—*

Contained :—

Nitrogen ... 0·1178 per cent. (by direct Kjeldahl).

Carbohydrate ... 83·3 per cent.

TABLE VI.

In the 24 hours ending at 10 a.m. on:—	Grammes of dry Carbohydrate consumed each day:—					Grammes of dry Fat consumed each day:—				
	As Milk.	As Biscuit.	As Cane sugar.	As Grape sugar.	As Arrowroot.	Total.	As Milk.	As Butter.	As Eggs.	Total.
Sept. 12	180.00	—	—	—	—	180	144.00	—	—	144
13	172.11	—	—	—	—	172	158.69	—	—	159
14	186.22	—	—	—	—	186	186.61	—	—	197
15	186.22	—	—	—	—	186	186.61	—	—	217
16	205.79	—	—	—	—	206	217.27	—	—	213
17	201.83	—	—	—	—	202	213.09	—	—	147
18	139.15	—	—	—	—	139	146.91	—	—	139
19	102.57	82.76	—	—	—	185	108.29	8.56	12.06	152
20	102.20	76.50	—	—	—	179	107.90	20.46	24.12	161
21	104.57	96.94	—	—	—	202	111.40	23.93	24.12	160
22	99.87	89.81	—	—	—	190	105.44	30.44	18.09	157
23	104.01	31.99	—	—	—	136	109.81	29.85	36.18	178
24	100.33	56.92	—	—	—	157	105.98	36.14	30.15	168
25	98.61	57.41	—	—	—	156	104.11	88.71	24.12	170
26	101.45	57.07	—	—	—	159	107.11	38.49	36.18	161
27	94.90	65.02	578.05	—	—	788	73.78	51.44	36.18	109
28	102.03	49.65	401.25	—	—	553	79.26	45.17	36.18	147
29	50.78	51.15	235.40	—	—	327	50.98	51.77	36.18	
30	75.90	50.92	236.80	—	—	424	58.97	51.77	36.18	

TABLE VI.—continued.

In the 24 hours ending at 10 a m. on:—	Grammes of dry Carbohydrate consumed each day:—						Grammes of dry Fat consumed each day:—			
	As Milk.	As Biscuit.	As Cane sugar.	As grape sugar.	As Arrowroot.	Total.	As Milk.	As Butter.	As Eggs.	Total.
Oct. 1	103.27	50.92	347.95	—	—	502	80.22	28.81	36.18	145
2	106.78	50.36	173.25	—	—	390	82.95	39.33	24.12	146
3	81.98	58.01	102.90	266.30	—	509	63.69	57.78	36.18	158
4	84.45	43.87	—	193.50	—	322	65.61	80.14	24.12	120
5	103.17	37.73	274.40	—	—	415	80.15	84.74	30.15	145
6	100.70	51.22	265.45	95.45	—	513	78.23	42.08	24.12	144
7	86.69	48.52	130.55	—	—	266	67.34	35.65	36.18	139
8	105.74	58.65	239.30	46.80	26.91	477	82.14	45.42	24.12	152
9	100.94	38.25	40.25	205.30	—	385	78.41	12.53	24.12	115
10	106.97	39.08	66.75	209.10	100.71	523	83.10	—	18.09	101
11	108.88	25.57	31.90	100.10	83.26	310	53.51	—	19.09	72
12	105.64	25.13	40.50	154.10	91.21	417	82.07	—	24.12	105
13	104.97	25.80	72.10	137.00	118.20	448	81.55	—	18.09	100
14	50.21	51.67	—	—	37.15	139	39.00	—	30.15	69
15	52.58	77.32	—	—	—	130	40.85	—	36.18	77



TABLE VII.

Blood-pressure in patient's radial artery, as recorded by an Oliver's blood-pressure gauge in mm. of mercury. Three independent readings in each case:—

		(A) Patient lying, arm horizontal, on bed, about 10 a.m. each day:—			(B) Patient standing, arm supported on pillows on a level with his heart; at about 6 p.m. each day:—		
		1st reading.	2nd reading.	3rd reading.	1st reading.	2nd reading.	3rd reading.
Sept.	12	160	150	160	—	—	—
	13	140	135	140	170	165	170
	14	140	135	140	160	165	160
	15	145	130	135	165	165	170
	16	150	140	140	150	155	150
	17	145	150	150	—	—	—
	18	140	150	150	150	155	150
	19	160	180	160	155	160	155
	20	145	140	140	175	170	180
	21	135	140	130	170	175	170
	22	140	140	140	175	175	175
	23	140	135	140	160	165	160
	24	140	140	140	155	160	160
	25	140	140	140	150	155	155
	26	135	140	135	160	165	165
	27	140	140	140	165	165	165
	28	135	140	140	150	155	155
	29	145	140	145	165	160	160
	30	140	140	140	170	165	170
Oct.	1	135	140	135	150	155	150
	2	150	150	150	165	170	170
	3	140	140	140	155	160	155
	4	145	140	150	150	150	150
	5	140	150	140	160	165	160
	6	140	140	140	165	170	165
	7	140	140	140	165	170	160
	8	135	135	135	165	170	170
	9	140	140	140	180	175	175
	10	135	135	135	180	175	180
	11	140	140	140	170	170	170
	12	140	140	140	175	180	175
	13	135	140	135	165	170	165
	14	140	140	145	180	180	180
	15	135	140	145	165	160	165

In regard to this Table, the writers would say that they found it extremely difficult to be sure of the point where the needle gave its maximum excursion. Moreover, they believe that the zero point of the instrument must have been too high, because normal students gave similar figures with the same instrument, and the figures are high. They regard the figures as of relative value only. (See page 145).



# THE IMPORTANCE OF STRONG ABDOMINAL MUSCLES.

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THE muscles of the abdominal wall, although anatomically insignificant, have several important functions to perform. From the point of view of physiology, the human body consists of the viscera enclosed in a shell, which varies in its properties in different parts; so that the box in which the brain lies is incapable of variation in size, that in which the spinal cord lies of slight variation, that for the thoracic viscera of considerable, and the muscular box, in which the abdominal viscera are situated, of by far the greatest variation. Since the brain substance is incompressible and nearly fills the rigid air-tight skull, it follows that the quantity of blood within the cranium must always be practically constant and that the only physiological vascular change which can take place within it, apart from those due to the heart-beat and the respiratory movements, is variation in the rate of blood-flow through the brain. This rate of flow must depend upon the difference between the blood-pressure in the carotid artery and superior vena cava; and it has been shown that variations in the venous pressure have a greater effect than do those in arterial pressure. In other words, the circulation through the most vital organ in the body is regulated, not by vasomotor changes in its own blood-vessels

but by alterations in the circulation through other organs, of which by far the most important are the abdominal viscera. An adequate cerebral circulation is only possible, therefore, as long as the abdominal circulation is properly regulated.

On its arterial side the abdominal circulation is regulated by the state of activity of the vasomotor centre, which, when in a state of increased activity, constricts the splanchnic arterioles and thus introduces into the circulation a vast resistance box which causes more blood to take the other circuits of less resistance, including the brain. On its venous side the splanchnic circulation is regulated by the respiratory centre, which by its action determines the activity of the respiratory pump, and by altering the tone of the abdominal muscles determines the size of the abdominal veins. Since these veins are readily distensible and could easily hold the entire circulating blood, the determination of their size by the abdominal muscles becomes one of the most important factors in maintaining the circulation.

During health the chief disturbing factor in the cerebral circulation would be the effects of gravity on the abdominal venous circulation unless these effects were promptly prevented. Anyone can prove for himself that the force of gravity can upset the cerebral circulation; it is only necessary, when tired or after an illness, to jump suddenly from the horizontal to the standing position to recognise in the giddiness, faintness, tinnitus and throbbing in the head that the circulation through the brain has momentarily failed. During health these effects of gravity are not produced, and that they are not is due to the perfection of the mechanisms which prevent them. These defensive mechanisms are: (1) acceleration of the heart, which should not be greater than about five or ten beats a minute. In various forms of ill-health, the heart may be accelerated by twenty or more beats a minute; this does not necessarily show that there is any weakness of the heart itself, but may simply mean that one of the other preventive mechanisms has partly failed, the heart has consequently to try to make up for this by a greater acceleration, which causes a greatly increased output of blood from the veins into the arteries. (2) Vaso-constriction of the

splanchnic arterioles by increased activity of the vasomotor centre; and (3) increased activity of the respiratory centre, whereby the action of the respiratory pump and the tone of the abdominal muscles is increased. In these ways more blood is pumped from the abdominal veins into the arteries, and by the increased splanchnic resistance determined to the head, so that the general arterial blood-pressure, and a proper rate of blood-flow through the brain are at least maintained. As a matter of fact, perfect compensation for changes in posture is of such vital importance to the proper working of the human body that a healthy man over compensates for the effect of gravity on his circulation and the blood-pressure in the brachial artery is actually slightly higher in the standing than in the horizontal position.

Perfect as these arrangements are in health, they cannot withstand the action of gravity for more than a certain number of hours on end. Death from crucifixion is an extreme instance of this. But even at the end of twelve hours or more, especially after severe muscular exercise, our bulbar centres begin to tire, we instinctively assume a more horizontal position, and by sighing and yawning increase the activity of our respiratory pumps, by stretching movements we compress our abdominal and other veins, and so force more blood into our arteries, and by sleep we completely restore the activity of our centres.

If, however, from illness or disease there is a partial failure of one of these mechanisms, then compensation may fail altogether, or if more or less complete at first, may last for a fewer number of hours than in health. To take a specific instance; supposing the abdominal walls are weak and slack, as long as the patient remains in the horizontal position the cerebral circulation may be normal; but directly she assumes the standing position, gravity will distend the abdominal veins with blood to a degree determined by the resistance of the abdominal muscles. If the distension of the veins is not too great an abnormal acceleration of the heart, combined with an increased activity of the vasomotor centre and of the respiratory suction-pump may for a time keep up a more or less normal cerebral circulation. But the

bulbar centres cannot work at such high pressure for long, and as they tire, the cerebral circulation will slacken and the patient feel fatigued. If the abdominal walls are sufficiently slack, it may be impossible for the patient to stand up at all without experiencing sensations of giddiness and fatigue.

That a want of resisting power in the abdominal walls really does have this effect upon the cerebral circulation, direct experiments have shown. For if a tame rabbit, which has a slack pendulous abdomen, be held up by the ears, it becomes unconscious in a few minutes, and dies in about half an hour from cessation of its cerebral circulation; it has, in fact, under the influence of gravity, bled to death into its own abdominal veins. A wild rabbit, which has a naturally firm abdominal wall, or a tame rabbit, whose abdomen has been firmly bandaged, if held up for the same length of time, can resist the action of gravity, and does not lose consciousness. In the same way, if a large accumulation of ascitic fluid be too rapidly withdrawn, the stretched abdominal walls are unable to contract up as fast as the fluid escapes, blood has to accumulate in the abdominal veins in order to fill the extra room in the abdomen so created, and the patient bleeds into his own abdominal veins.

We thus see that strong abdominal walls are essential to an active life in the feet-down position; and that, if they are weak, according to their degree of weakness the patient must suffer from symptoms intermediate between merely feeling tired more rapidly than normal people, and being unable to stand up at all without symptoms pointing to a failure of cerebral circulation.

We have now to ask, supposing a patient from weakness of her abdominal walls suffered from chronic failure of the cerebral circulation, what symptoms should we expect her to exhibit? In the first place, we should expect that whatever the symptoms were they would be greatly relieved by lying down, and that a patient finding this out for herself might easily drift, from want of proper treatment, into a condition of chronic invalidism in the horizontal position. In the second place, we should expect some of the following symptoms in a greater or less degree, and after a shorter or longer period in the feet-down position, headache,

vertigo, tinnitus, and other sensations in the head, mental irritability and insomnia, or mental dulness and somnolence; deficient memory of recent events, indecision, and a disinclination to do anything requiring an effort; muscular weakness and a rapid onset of fatigue. On examination we should expect to find badly filled arteries, a rapid, irritable heart, and a deep cardiac dulness outside the nipple line. The patient would certainly complain of loss of appetite and other symptoms of primary gastric insufficiency, by which is meant failure of digestion, owing to failure in the secretion of gastric juice, the failure of secretion being due primarily, not to the stomach, but to the brain. For it has been shown that the mere presence of digestible food in the stomach does not of itself produce more than a slight, temporary and totally inadequate secretion of gastric juice. In order to get the prolonged secretion required for the digestion of a meal, it is necessary that peptones should first be absorbed by the mucous membrane of the stomach. The question naturally arises, where is this peptone to come from? The answer is that when we see or smell food, and directly we begin to masticate it, a secretion of gastric juice is set up reflexly through the brain, so that when food reaches the stomach it is met by gastric juice already secreted, peptones are quickly made and absorbed, and a prolonged secretion of gastric juice ensured. It is found that the absorption of the nitrogenous extractives of meat have the same effect as peptones. For this reason, we begin a big dinner with soup, in order to prompt the stomach with meat extractives and a readily peptonisable food. If, however, through illness, or tiredness of the brain, the early reflex or psychical secretion fails, the whole of gastric digestion must fail, or be much prolonged. Everyone, when over-tired, has experienced complete loss of appetite, and the fact that if a heavy meal be eaten it will probably be vomited undigested in a few hours' time. Under the circumstances, the wise man, instead of eating, lies down and goes to sleep; or if he does eat, he begins with a dose of meat extract or peptone.

It may well be asked, what does it matter if a patient always does have prolonged gastric digestion; the stomach is a mere receptacle for food, it absorbs nothing except small quantities

of peptones and when excised the patient's powers of digesting and absorbing food are not diminished. But unfortunately the stomach possesses a pyloric sphincter which allows scarcely any food to leave the stomach until most of it has been broken down to a certain small size; therefore insufficient secretion of gastric juice must mean a prolonged stay of solid food in the stomach; this in turn, sooner or later, means acid fermentation of the food and the painful and unpleasant symptoms of gastric irritation.

We should expect, then, to find, and we certainly do find, that weakness of the abdominal walls does lead to a number of symptoms similar to those enumerated above. These symptoms are not infrequently accounted for by calling them functional, neurotic or hysterical; or the patient is called a neurasthenic, and then does the rest cure with or without seclusion from sympathising friends, or undergoes a course of dieting and drugs, and last, and by no means least, as we shall see, laparotomy.

Anyone who has compressed with his hand the abdomen of one of these patients and felt the radial artery instantly fill up with blood and the pulse-rate drop, or has made the patient jump from the horizontal to the standing position with and without a firm abdominal bandage, counting the pulse and noting the difference in the patient's subjective symptoms in the two cases, will have come to the conclusion that the rational and only effectual treatment is to strengthen the abdominal walls. This can be done in two ways. The abdominal muscles may be strengthened by gymnastic exercises. It is only necessary to know the movements of the body in which the abdominal muscles are used in order to be able to devise exercises to bring them into play. In all cases of weak abdominal walls exercises are of use; but when the abdominal muscles have been greatly overstretched or the recti are widely separated, exercises cannot bring back the abdominal walls to their normal condition. By firmly bandaging the abdomen, however, the capacity of the abdominal cavity can be reduced to its proper size and the function of the abdominal muscles in maintaining the cerebral circulation to a large extent replaced.



Next to regulating the size of the abdominal veins, the most important function of the abdominal muscles is to support the abdominal viscera. Some anatomists describe these viscera as held in position by certain ligaments, folds of the peritoneum, and even by their blood-vessels. It is only necessary to compare the organs with the thin and very extensible materials which are said to hold them in position, to see that this can hardly be the whole truth. In the standing position the weight of the viscera is borne by the abdominal walls, and if they are strong enough to withstand it, the viscera do not drop simply because there is no space into which they can descend. If, then, the abdominal muscles are strong, the abdominal cavity in the feet-down position remains the same size, and the viscera as a whole are kept up. The function of the various visceral attachments is to prevent the viscera from changing places amongst themselves and to keep them in the same relative position to each other.

If, however, the attachments of any viscus are abnormally weak or slack, a strong abdominal wall will not prevent gravity from causing a change in its position relative to the other viscera. As instances of this it is only necessary to mention some forms of movable or floating kidney, wandering spleen, floating liver, the displacement of a loaded or dilated stomach, and the displacement of an isolated viscus caused by the contraction of adhesions. None of these displacements necessarily cause symptoms, but, if they do, it is clear that, when the abdominal walls are strong, no strengthening of those walls nor bandaging of the abdomen can do anything to prevent the viscus moving about.

When the abdominal walls are so weak that they can no longer withstand the weight of the viscera in the feet-down position, we have a totally different condition to deal with. The viscera as a whole, it may be some more than others, begin under the force of gravity to drop down and in so doing they pull on and stretch their various attachments producing the condition known as general abdominal ptosis, visceroptosis, enteroptosis or Glenard's disease. The effect that an abdominal wall in this condition has upon the cerebral circulation and the symptoms

which ensue, have already been dealt with; it is only now necessary to enquire what additional symptoms we should expect to be caused by the visceroptosis itself. The chief one would certainly be various dragging and other uncomfortable sensations in different parts of the abdomen, also pain caused by the dragging on and stretching of visceral nerves. Various physiological researches have taught us that abnormal afferent impulses starting from the viscera can reflexly upset the proper working of many organs, for instance, the heart. We also learn that these impulses would be likely to interfere with the secretion of the gastric and pancreatic juice, produce vomiting and restrict the movements of the stomach and intestines.

Further, besides the abdominal pain, we should expect in many cases to find pain of the reflected visceral type; that is, afferent impulses from an affected viscus passing up the sympathetic and reaching the corresponding segments of the spinal cord, so modify these segments that, when afferent impulses from other structures reach them, pain and tenderness in corresponding segments of the body are produced. It has been recently shown that if this reflected visceral pain is of sufficient duration and intensity, certain mental changes may be, and are often set up, which are still generally considered to be hysterical. These mental changes include a sense of ill-being, fits of depression and suspicion, visual, auditory or olfactory hallucinations, failure of attention and memory of recent events; they are more easily set up in women than in men, and are completely outside the control of the patients. It is almost unnecessary to point out the effect that chronic failure of the cerebral circulation would have upon the liability to these attacks. We are in health so oblivious of the presence and workings of our viscera that when, from disturbance of them they obtrude themselves upon a patient's consciousness, it is not surprising the whole of the patient's attention should be rivetted on her various abdominal sensations. These so-called intensely neurotic patients are generally so, only because of their visceral disease, and will remain so until that is cured. If, then, after treatment, whether medical or surgical, the pain and other symptoms return, the

fault probably lies in the treatment, and not necessarily in the patient's mental state, as is so often assumed.

We should also expect that the various symptoms would be considerably relieved, if not altogether absent, in the horizontal position.

Cases of abdominal ptosis have been divided into three clinical groups :—

1. Those in which it exists, but without producing symptoms appreciable to the patient. The case cited in support of this is that of an unmarried lady of 52, all of whose viscera were prolapsed; but she complained only of an abdominal tumour which proved to be a normal liver, and which, when she stood up, reached the level of the umbilicus. Her general condition is described as follows :— “ The case was a pronounced example of visceroptosis; yet the patient had no abdominal symptoms of any kind. Her digestion was good, and her bowels regular. She was a placid person, who lived at her ease, and was at peace with herself and all men. She stated that she was not strong, but that she lived carefully and enjoyed excellent health. The ‘tumour’ had disturbed her in no way, and had merely aroused an amused curiosity. She was feebly pleased to hear that she could continue to take carriage exercise.” In spite of the absence of abdominal symptoms, it would be hard to maintain that such a patient was physically and mentally normal. It is possible to state, without fear of contradiction, that no one with a weak abdominal wall untreated can attempt to lead a normally active, much less an arduous life, without showing bodily and mental symptoms, due to failure of their cerebral circulation. All that patients like the above one show is that it is possible to have marked visceroptosis and consequent dragging on the visceral attachments, without any abdominal pain. How this is possible we need not wait to discuss. All that it is necessary to point out is that a patient with visceroptosis which causes her no pain, none the less requires treatment for the condition of her abdominal walls, and that, unless she receives it, she can only be expected to lead, in comfort, a life similar to that of the above patient.

2. Cases of abdominal ptosis "which may be attended by a definite series of nervous symptoms, which may be distressing and even alarming," and we are gravely asked to believe in regard to these symptoms "that they may be made to disappear by a measure of treatment (abdominal incision) which apparently can act through the nervous system only." The case quoted in support of this opinion is that of an unmarried lady of 80, who was feeble, anæmic, and intensely neurotic. For ten years she had suffered from agonizing abdominal pain which health resorts and medicine had failed to cure. The abdomen was flabby, both kidneys slightly movable, and there was a moderate degree of general ptosis of the abdominal viscera. Since all medicines had failed and the patient was a chronic invalid, the abdomen was opened. General prolapse of the viscera was found, but all the organs were perfectly normal. The exploration cured the patient of all her symptoms, and we are further told:—"This case therefore was one of uncomplicated visceroptosis with nervous phenomena in a very neurotic patient. Although the patient had never worn a belt, she was always worse when she moved about, and was only comfortable when in bed. A belt was worn after the operation." This case and its cure is clearly open to a totally different interpretation. It may be taken to prove that such a patient is neurotic only because of the visceral pain due to the visceroptosis and that if the latter is removed, by supporting the abdominal wall, the pain and the neurosis both disappear. To refer to these patients as "those in whom a neurotic tendency exists and in whom the mobile viscus becomes the focus of a number of indefinite and bizarre nervous symptoms" is surely to mix up cause and effect, and to ignore the direct connection between visceral pain and certain mental states.

3. Cases in which the abdominal ptosis depends upon adhesions dragging the various viscera downwards towards the pelvis. This downward movement of the viscera is only possible when either the contraction of the adhesions is able to overcome the resistance of a normal abdominal wall or the abdominal muscles were previously weak. In either case pain will be caused chiefly by

the stretching of ligaments and other normal attachments of the viscera and will be only to some extent relieved by the horizontal position or by wearing an abdominal bandage when in the upright position. It may be difficult, but it is important, to recognise this condition clinically, since surgery offers the only chance of relief. Its presence may certainly be suspected under the following circumstances: When attacks of visceral pain still come on in spite of a horizontal position; when any or all of the displaced viscera seemed fixed in their abdominal position and cannot be replaced easily when the patient lies down. This suspicion would be greatly strengthened by a history of ulceration of the stomach or intestines or of tuberculous or other inflammation in the abdomen or pelvis. If the adhesions are divided the patient may be relieved of all pain, but she may not; and here again the moral to draw is not that she is an incurable neurotic, but that the treatment has been incomplete. For, when the adhesions have been divided, the general visceroptosis may still exist, dragging on the visceral attachments may still take place and be sufficient to cause pain and keep up the "neurotic" state.

Another class of case is that in which adhesions round such an organ as the stomach complicate but are not the cause of visceroptosis. Here pain may be caused by dragging on adhesions as well as on the normal visceral attachments. The following is such a case:—A married woman of 35 had had the symptoms of gastric ulcer on and off for twelve years, including occasional attacks of hæmatemesis. Her chief complaint was of abdominal pain situated in the epigastric angle; some pain was more or less constant but it increased in paroxysms which rarely came on until two hours after food, and might come on when no food had been taken. These paroxysms were sometimes sufficiently severe to require morphia. Complete rest in bed, with rectal feeding for a fortnight, did not completely remove these paroxysms, so the abdomen was explored. At the operation adhesions between the stomach and liver were found and divided, the pain disappeared and the patient returned home apparently cured. At the end of three months of more or less hard work

at home, constant epigastric pain again appeared and the patient rapidly developed the former state of ill-health and her neurasthenic symptoms. Three weeks in bed on an ordinary diet caused the pain and other symptoms to disappear, and the patient was going to be discharged as an incurable neurasthenic, when it was pointed out that she had extremely slack abdominal walls. Examination in the standing position further showed that she was suffering from well marked visceroptosis. She was accordingly advised for the rest of her life to bandage her abdomen firmly before getting out of bed in the morning, and, if during the day the bandage became displaced, to take it off and replace it when in a lying position. At the end of a year she was still able to do her work in comfort, and although not a strong woman, she was at any rate far from being an invalid.

In the same way it happens not infrequently that a patient complaining of abdominal pain is found to have a movable kidney. The kidney is stitched and secured in its proper position, and, as long as the patient remains in bed after the operation or leads the quiet life of convalescence or wears a belt, there is no return of symptoms. But, when the belt is discarded and an active life resumed, the patient again becomes neurotic and the pain returns. The case is then often considered to be one of confirmed neurasthenia and the return of the pain is accounted for by the mental condition. A large number of such cases clearly can bear quite a different interpretation and the sequence of events in them is as follows, namely, stretched abdominal walls, a poor cerebral circulation, general visceroptosis of which the movable kidney was but a part; the improvement was caused not by the operation but by the enforced rest and abdominal support, and, as soon as these were removed, back came the pain and the mental symptoms due to it.

Several circumstances combine to render visceroptosis easily overlooked; of these the most important is the habit of examining the abdomen only with the patient only lying on her back and not in the standing, knee-elbow, or some other similar position as well. In this way the condition of the abdominal

muscles and the mobility of the viscera is often overlooked ; and without a knowledge of these necessary data it is impossible to apply the proper treatment. For, if we find that the abdominal muscles are of normal strength and unstretched, we know that general ptosis of the viscera cannot exist. If in such a case there is abdominal pain and a kidney or other organ is found to be movable, and if there is reason to believe that the movable viscus is causing the pain, then the treatment is surgical and will probably cure the pain permanently. When, however, the abdominal walls are slack we know that general visceroptosis is to be expected. If in such a case there is abdominal pain and we find a movable kidney, we do not know that the kidney more than any other of the viscera is the cause of the pain, and to sew the kidney alone would be absurd, unless it were causing some trouble mechanically.

Another reason why prolapse of the viscera as a whole is easily overlooked, is the fact that we have no clinical method of determining the exact position of the organs first and most frequently prolapsed in visceroptosis, namely, the colon and small intestines ; even the stomach presents difficulties to many unless auscultatory percussion is used. Consequently the position of only the solid organs is generally determined because this can be ascertained by palpation ; and of these organs the only ones which are commonly much prolapsed are the kidneys. In well marked cases of visceroptosis the condition can be diagnosed at a glance, for, when the patient stands, the upper part of the abdomen becomes flattened and the lower part is bulged forward by the weight of the prolapsed viscera.

A not unimportant point to determine is, how far the site of the pain in the abdomen and of the skin tenderness can be used to determine which of the abdominal viscera is causing the pain. It may be said at once that the pain may give very little help as a localising symptom. When a kidney is dragging on its attachments the pain may be confined to the corresponding loin, but it may also radiate widely over the back and abdomen. Ptosis of the intestines and stomach generally causes pain, and a dragging sensation to be complained of at some point near the middle line,

between the epigastric angle and umbilicus, and this point in many cases seems to correspond roughly to the situation of the celiac axis or the inferior mesenteric artery and nerves. Pain and tenderness at these points might, however, be due to so many other conditions as to be of little localising importance. With regard to skin tenderness, we get but little help by mapping it out, for the segmental supply of the viscera overlaps to such an enormous extent that skin tenderness in one or more segmental areas might be due to any or all of several viscera.

Enough has been said to show that strong abdominal muscles are of importance to the human body. The bearing which this fact has upon the treatment of the abdominal walls during and after laparotomy, and after the over-stretching of the abdominal muscles by pregnancy, ascites, ovarian tumours, fat, etc., is too obvious to need mention.

With regard to the frequency of weak abdominal walls, and of the symptoms arising therefrom, it is no exaggeration to say that it is comparatively rare to find amongst out-patients a multiparous woman with abdominal walls of a strength approaching the normal; it is also no exaggeration to say that a large proportion of the married women who crowd the medical out-patient department of a hospital for various subjective and objective symptoms of ill-health, over-work, neurasthenia, indigestion, etc., are suffering from little else than the results of the weakness of their abdominal walls. This condition is by no means confined to women of the poorer classes nor to married women. Although less common it is by no means rare amongst flabby and anæmic unmarried women, but it is extremely uncommon in men. Into all the possible causes in an unmarried woman it is unnecessary to go, but this difference between unmarried women and men is striking, and its probable explanation has a bearing upon the treatment of the condition when it does arise. It seems probable that the wearing of corsets is a not unimportant cause. Not only do they, by constricting the upper part of the abdomen, force the abdominal viscera downwards and so put a constant extra strain on the abdominal walls below; but by making thoracic instead of abdominal respiration



necessary must lead to the partial atrophy from disuse of the abdominal muscles. Therefore, in treating this condition it is necessary not only to support firmly the lower part of the abdomen, but also to exercise the abdominal muscles by gymnastics and to make abdominal respiration, which is just as much normal to women as men, possible, and to see that the patients learn to use it.



# SOME PATHOLOGICAL ALTERATIONS OF THE IRON IN THE LIVER.

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THE most familiar and striking instances in which the iron in the liver is altered, both in amount and in the nature of its combinations, are found in cases of pernicious anæmia.

Besides alteration in the size and colour of the organ, it can be readily demonstrated in the majority of cases by means of Perl's test that certain profound changes have occurred in the iron stored in the viscus. The way in which these changes are brought about is still very obscure, and it may be possible to throw some light on it by investigating other conditions in which similar changes take place.

It would naturally occur to anyone that, if iron were absorbed from the intestinal tract, the portal blood would carry it to the liver, and there store it temporarily, so that in cases of hæmorrhage, where the blood was passed down the intestines, one would expect to find an excess of iron in the liver.

Abderhalden (*Zeit schr. f. Biologie*, vol. xxxix., p. 14) says that iron can be absorbed by the small intestine from hæmatin and hæmoglobin, and cases 4 and 5 in this paper are pathological examples of this fact.

I have been able to test a great many livers from autopsies performed at Guy's Hospital during the last two years by Perl's test, and in some of the cases Mr. H. Williams, B.Sc., has very kindly estimated the percentage of iron in the organs.

In the investigation of such a question as the pathological changes of the iron in the liver, much more elaborate methods seem to me to be necessary than I have had the time for ; so this must be my excuse for the incompleteness of this paper.

Hopkins (Five cases of Pernicious Anæmia, *Guy's Hospital Reports*, vol. L.), says that there is no general correspondence between the intensity of the Prussian blue reaction obtainable in any case and the actual percentage of iron found, and concludes that this fact is an indication that the hepatic iron in pernicious anæmia may vary a great deal as regards the stability of its combinations.

He goes on to say that the actual percentage of iron in even a normal liver would probably be ample to yield a Prussian blue colour were it present in compounds of sufficiently loose type.

From my own investigations I found that it was certainly quite exceptional to get this reaction in livers taken from a great variety of diseases. It seems, then, to be safe to conclude that when this reaction is obtained there is something abnormal either in the quality or quantity of iron in the organ.

The test, as I have employed it, consists in placing thin slices of liver, which had been previously gently washed in water, in a 5 per cent. solution of potassium ferrocyanide for three minutes, and then immersing them in a 1 per cent. solution of hydrochloric acid. The blue colour should be quite manifest at the end of one minute in this latter reagent. A very long immersion in the acid produces the reaction in most livers, so that it is important to obtain it quickly. If the hæmolysis in pernicious anæmia were dependent on the anæmia, one would expect to get this change in the liver in other diseases associated with a severe anæmia ; but this is not found to be the case.

Dr. Hunter has advanced the theory that in pernicious anæmia there is a septic infection of the alimentary tract which produces hæmolysis in the portal blood. If this is so, then it might be expected that other septic states of the mouth or stomach would produce similar changes in the portal circulation.

I think cases 1 and 2 show that under certain circumstances this does occur. In the first of these the patient suffered from an

ulceration of the tonsils followed by an abscess around the knee-joint, and died with all the evidences of a severe septic poisoning. A few days before death he became very anæmic, of a yellowish tint somewhat resembling that seen in pernicious anæmia. At the autopsy the blood-clots were extremely pale, and the liver had the pale fatty look often seen in that disease. A careful enquiry into his history before admission quite negatived the idea that he had been suffering from a primary anæmia before his last acute illness, and I think that there can be no doubt that the above changes, as well as the striking alteration in the iron in the liver, as shown by Perl's test, were due to a virulent infection by organisms which obtained an entry into his system through the tonsils.

In case 2 the periosteum of the upper jaw was the starting point of the disease, and led to a very severe septicæmia. In both these cases organisms must have been swallowed, and then probably set up in the alimentary tract changes in the portal blood, which led to alterations in the iron contained in the liver.

In these two cases I was unfortunately unable to have the organs analysed. In two other instances in which the patients died from acute septic affections of the mouth I did not obtain the reaction, so that it seems possible that some particular organism is responsible for the change.

In case 3 there was an oozing of blood from the gums for some days before death, but this never amounted to very much. Possibly it may have been sufficient to allow the intestinal tract to absorb iron from it after it had been swallowed, but I am much more tempted to explain the change in the liver as consequent on a septic process originating in the mouth, for this was in a very unpleasant condition.

In cases 4 and 5 the iron was doubtless absorbed from the gastro-intestinal tract during life, but in other instances where similar hæmorrhages had occurred no reaction was obtained, so that it would seem that either the iron was for some reason in more stable combination, or that there were differences in the intestinal mucous membrane affecting its absorption. Possibly such differences may be dependent on bacterial action.

In case 6, one of acute hæmorrhagic pancreatitis there was certainly some blood extravasated into and around the pancreas, but its total amount was small, and I find it difficult to imagine that iron was absorbed from it in sufficient amount to give such a pronounced reaction. In this disease there is no doubt that changes take place in the portal circulation, and the frequency of the occurrence of jaundice shows that the liver must be involved, so that hæmolysis of the portal blood, and not mere absorption of iron from the pancreatic tissues may be the explanation of the change in this case.

Case 7 was one of severe erysipelas of the face. The patient had complained of a sore throat, and the fauces were said to be infected. The condition of his face was such as to make it difficult to exclude the septic mischief from his mouth. Possibly here there was an infection of the gastro-intestinal tract. The only other way to explain the change in the liver would be to say it was caused by a toxin circulating in the blood, but I did not get the reaction in three cases of erysipelas of other parts of the body. The percentage of iron in the liver in this case was more than double the normal.

Dr. Maude Abbott, in a description of a case of pigmentation cirrhosis in hæmochromatosis (Path. Soc. Trans., vol. li.), mentions five cases in which there occurred an iron-containing pigment in the liver, all of which, except one, reacted well to Perl's test. In all of these cases there was a history of intestinal disturbance, and either acute inflammation of the wall of the gut, venous congestion, duodenitis, or hæmorrhage.

She goes on to say that hæmosiderosis was observed in her cases (as in many in the literature) to be associated with a history of intestinal disturbance, and at times, of prolonged suppuration. I have tried the test in a number of instances of chronic suppuration, and in many of acute septic disease, such as acute epiphysitis, pelvic septicæmia, suppurative peritonitis, etc., but have not obtained it.

It will be seen that Mr. Williams found an increase in the percentage of iron in the liver in all the cases submitted to him. A normal liver he found to contain .089 per cent. of iron, which

closely agrees with the normal as given by Hopkins (*loc. cit.*), *i.e.*, .09 per cent.

The following is the method adopted by Mr. Williams in estimating the amount of the iron in the organs :—

The liver was first preserved for a few days in formalin. This was proved to be practically free from iron by evaporating 200 c.c. to dryness, and testing the very small residue with hydrochloric acid and potassium ferrocyanide. Only minute traces could be found.

The organ was then washed as free from blood as possible, and cut into small pieces, in case 5 by hand with a razor, but in all the other cases by means of a carefully cleaned sausage machine, which produced a much finer state of sub-division.

The finely divided organ was then washed in running water for five to eight hours, after which time the water which left the liver was quite colourless, except for small pieces of liver carried away. The organ was then dried at 100° C. for three days, as experiments on the first liver tested showed that at the end of this time the weight was constant.

For the determination of iron (Hopkins' method) 10 grammes of liver were accurately weighed and ashed in a platinum basin, and not in a muffle, as described by Mr. Hopkins, but over a Bunsen burner, this having less tendency to fuse the ash. The clean white ash was then dissolved in dilute H.C.L., and evaporated to complete dryness on a water-bath with a few drops of strong nitric acid to oxidize the iron. The residue was re-dissolved in H.C.L. and precipitated with dilute ammonia. The precipitate, filtered off and washed, was re-dissolved in dilute sulphuric acid and reduced with zinc. The resulting liquid was then titrated with centinormal permanganate. The zinc was carefully tested and found free from iron.

Two complete determinations were made in each case and the mean taken. The following are the results :—

No. of case.	Ash.	Percentage of Iron.
4 ... ..	3.1 ... ..	.136
5 ... ..	3.0 ... ..	.25
6 ... ..	— ... ..	.134
7 ... ..	— ... ..	.22
Normal liver ... ..	... ..	.089

I have concluded, then, that the essential factor in the production of an alteration in the iron stored in the liver is hæmolysis of the portal blood, and that this is dependent on gastro-intestinal processes probably of a bacterial nature. In cases of hæmorrhage where direct absorption may take place the effect on the liver as judged by Perl's test is not constant.

Sections of the livers treated with the cyanide salt and hydrochloric acid showed the iron to be almost entirely in the liver cells.

#### SHORT ABSTRACTS OF CASES.

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CASE 1.—M. S., male, æt. 40. Admitted under Mr. Symonds into the hospital on May 2nd, 1900. Had been quite well until ten days before admission, when, in common with many of his family, he had a bad throat. His knee began to swell a few days before admission and was acutely painful. Was sent to the hospital, where on admission he was dyspnœic. Temperature 103·4°, pulse 120, respiration 40. Knee much swollen, hot and tender. Tonsils ulcerated. Free incisions made on either side of patellar and six ounces of pus escaped. May 3rd, 10 c.c. of antistreptococcin injected. He got much worse, became very anæmic and died May 5th.

*Abstract of autopsy.*—Large fat man. Anæmic, of yellowish tint; no jaundice. Lungs congested. Heart fatty, otherwise normal. Tonsils: on each side there was a deep ulcer and on section the tonsils contained thick yellow pus. No cellulitis of adjacent structures. *Stomach.*—Hæmorrhagic erosions. *Intestines*, normal in appearance. *Liver.*—Large, pale and fatty; reacted strongly to Perl's test. Bile, thin and pale; duct patent. *Spleen*, normal; gave Perl's test. *Kidneys*, normal; did not give test. Some of the pus removed at the operation was sent for bacteriological examination, but unfortunately got mixed with a little lysol and no cultures could be made.

CASE 2.—Richard B., æt. 35. Admitted under Dr. Pitt into Stephen, on May 12th, 1901, for ulceration and sloughing of



mouth. Is a plumber by trade. On May 3rd, had an abscess beneath right lateral incisor of upper jaw. May 5th, had expistaxis.

*Condition on admission.*—Temperature 103°, pulse 128, soft, respiration 30. Greyish membranous stomatitis affecting upper lip, and extending back as far as hard palate. Slight albuminuria. Apical systolic bruit. May 14th, temperature 104°, pulse 120, respiration 24; enlarged gland on left side of neck. May 17th, very anæmic; slough spreading backwards; had rigors. Died May 18th.

*Autopsy.*—Was only partial. *Heart.*—No endocarditis. Small hæmorrhages beneath pericardium. Fibrinous clot in the heart which had a most peculiar greenish yellow colour. *Liver.*—Pale, otherwise normal in appearance. Reacted strongly to Perl's test. Bacteriological examination of sloughs in mouth shewed presence of streptococci and staphylococci.

CASE 3.—F. E., male, æt. 9. Admitted under Dr. Hale White on July 7th, 1900, for pyrexia and pains in joints. Had had rheumatic fever. Had a sore on heel for ten days previous to admission, which caused a swollen gland in the groin. July 4th, had sudden vomiting with pain in limbs and stomach.

*Condition on admission.*—Ankle-joints swollen. Dulness with bronchial breathing at left base. Temperature 102°, pulse 120, respiration 40. Urine 1084; dark; slight trace of albumen. July 10th, much better. July 16th, temperature has kept up, but general condition improved; teeth covered with sordes. July 17th, spleen can be felt. July 20th, is very bad; chest and abdomen covered with a papular rash. July 23rd, temperature keeps up; enlarged glands behind ear and in neck. July 24th, wrist swollen. July 26th, general condition very bad; mouth foul, teeth covered with sordes. July 27th, bleeding from gums and nose; antistreptococcin injected. July 28th, slight leucocytosis (coarsely granular eosinophiles). Temperature 103·6°. Died.

*Autopsy.*—Œdema of aryteno-epiglottic folds. Tonsils normal. Old pleurisy over both lungs. *Spleen* large and congested, 170

grms. *Liver* 660 grms; pale; reacts well to Perl's test. Other symptoms normal. Some blood taken from the heart for bacteriological examination yielded two kinds of bacilli in great quantities, but on further growth they could not be differentiated and it was impossible to identify either.

**CASE 4.**—Mary G., æt. 58. Admitted under Dr. Hale White September 26th, 1901, for hæmatemesis. Died September 30th, 1901 (H. W. 814, 1901). Family history, unimportant. Has suffered much from rheumatism and dyspepsia. During last three weeks has complained of acute stabbing pain in abdomen. September 23rd, sick twice, bringing up blood. September 24th, hæmatemesis. This continued up to admission, blood being passed per rectum.

*Condition on admission.*—Blanched and too bad to be examined. Temperature was raised 100° to 101°, and on September 30th, rose to 102°.

*Post-mortem.*—Ulcer on posterior wall of stomach. Opening small vessel. Colon full of blood. Other systems normal.

**CASE 5.**—George H., æt. 29. Admitted under Dr. Washbourn, August 15th, 1901. An ordinary, but somewhat severe case of enteric. Temperature kept at about 103°. Had three severe hæmorrhages on August 26th, and died on August 27th.

*Post-mortem.*—Typical ulceration of intestine, which contained blood. Liver normal in appearance; gave good reaction with Perl's test.

**CASE 6.**—James H., æt. 51. Admitted under Dr. Shaw September 10th, 1901. Had been quite well until September 8th, when he began to vomit and had abdominal pain. Vomit became stercoraceous on September 10th. Bowels not opened.

*Condition on admission.*—Abdomen distended, skin sallow but no jaundice detected by gaslight. Was operated on at once, but died on the table.

*Autopsy.*—Fat necrosis around pancreas, which contained hæmorrhages into its tissues. No hæmorrhages elsewhere. Systems otherwise normal. Conjunctivæ jaundiced. Greyish,

shiny liquid in bowel, looking like pus. Liver normal to naked eye; reacted well to Perl's test; microscopically showed fatty changes.

CASE 7.—William S., æt. 34. Admitted December 4th, 1901, for erysipelas of face. Had been drinking heavily lately. On December 3rd, had a sore throat and redness about face. Several rigors, and had difficulty in swallowing.

*Condition on admission.*—Temperature 108°. Eyes and both cheeks and forehead showed marked erysipelas, with some blebs. December 7th, delirious; mouth wash ordered. December 8th, temperature 106·4°; died.

*Autopsy.*—Lungs congested. Old pleurisy. *Liver.*—Normal appearance, 1870 grms.; marked reaction to Perl. Other systems all normal. Bacteriological examination showed presence of streptococci and staphylococci in serum taken from the blebs.



# ON A NEW METHOD OF OBSERVING PEPTIC ACTIVITY.

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(GULL RESEARCH STUDENT.\*)

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1. Introduction.
2. Method of working.
3. Experiments.
4. Action of hydrochloric acid without pepsin.
5. Action of pepsin and hydrochloric acid.
6. Comparison of curves obtained by treating syntonin solution with differing proportions of pepsin.
7. Note by Dr. Wade.
8. Summary.

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## 1.—INTRODUCTION.

THE rapidity of proteolytic digestion is usually estimated by observing the rate at which a solid piece of proteid is converted into soluble proteid in the presence of the ferment, and under

\* This research was published in Germany in the *Heitschrift für Physiologische Chemie* for July, 1902.

the conditions favourable to its action.<sup>1</sup> The polarimetric method suggested by Schütz and Huppert<sup>2</sup> is certainly available for proteid in solution, as is the spectrophotometric method of Klug,<sup>3</sup> but as in both of these the early products of digestion must first be removed, they do not offer a means of continuously observing the transformation.

It was therefore suggested to the writer by Professor Kossel that possibly the progress of digestion in a solution of proteid was accompanied by a change in the viscosity of the fluid, and that if this were the case, such a change might furnish data by which the process could be observed without the labour of chemical manipulations.

The following investigation was accordingly made in the physiological laboratory at Heidelberg, and it is at the same time the pleasure and the duty of the writer to record here his deep sense of gratitude to Professor Kossel for the use of the laboratory, for his constantly displayed interest, and for the suggestions and advice received from him in the course of the experiments.

Little attention has been paid to the viscosity of proteid solutions other than blood.<sup>4</sup> The subject has, however, been worked at by Bottazzi,<sup>5</sup> who examined various physiological fluids and solutions, and made comparative estimations of their viscosity. He showed, among other things, that a peptone solution is less viscous than one of casein of the same strength.

<sup>1</sup> As in the well known methods of Bidder and Schmidt, Grünhagen, Grützner, and Mett.

<sup>2</sup> Pflüger's Archiv. Bd. 80, S. 470, 1900.

<sup>3</sup> Ungar. Archiv. f. Medizin Bd. 3, S. 87, 1895.

<sup>4</sup> The literature of blood viscosity is given by Hürthle, Pflüger's Arch., Bd. 82, S. 415. See also Trommsdorf, Arch. f., Exp. Path. u. Pharmac., Bd. 45, S. 66, and Hirsch and Beck. Münch. Medic. Wochenschr, 1900, S. 1685.

<sup>5</sup> Archiv. Ital. de Biolog., Bd. 29, p. 401.

## 2.—METHOD OF WORKING.

### DESCRIPTION OF APPARATUS.

The viscosity of the fluid to be digested was measured by means of the Ostwald viscometer,<sup>6</sup> the form of the apparatus being adapted from that used by Hirsch and Beck<sup>7</sup> for the estimation of the viscosity of human blood.

The following figure shows the actual arrangement :—

FIGURE 1. (p. 200.)

A is a large bottle standing on a shelf and containing water; this bottle is connected by a tube, part of which is india-rubber and part glass, to the lower part of another large bottle, C, which contains a little water. C is supported by a clamp to an iron stand, on which, by means of another clamp, it can be raised or lowered at will. In this manner the required pressure is produced. The neck of C is closed by an india-rubber stopper, through which a tube, D, passes, leading the air now under pressure, to the calcium-chloride tube, E; the other limb of the latter is connected with the pressure flask, F. From F the tube G leads to a T-piece, H, from which the tubes I and J lead off. I is joined to the manometer, K, filled with benzol of a specific gravity of .735 at 13.5° C. The difference between the level of water in A and that in C was so adjusted that a pressure was produced of 450 mm. of benzol, and this pressure was employed throughout. The tube J leads to the viscometer; it is provided with a spring clamp, K, and between the clamp and the viscometer is a glass junction piece, L.

The Ostwald's viscometer used is shown in detail in Figure 1, on the left. It consists of a reservoir, a, bounded above by a constriction, b, and below by the upper opening of the capillary tube, c. The glass tube into which it opens below is curved in U form, and then dilates into the small bulb, d. Just above d is a ground joint, into which fits the vertical tube, e.

The viscometer is immersed in a water-bath, N, and is supported on a separate stand, so that it can be taken in and out of the bath at will, without disturbing any other part of the apparatus. The water-bath is also provided with a stirrer, O, a thermometer, P, capable of being read to tenths of a degree; the bulb of a mercury gas regulator, Q, and a glass vessel, R, containing a supply of the same solution as is under examination in the viscometer.

<sup>6</sup> Ostwald. *Phys. Chem. Messenegen*. The viscometers used were made by C. Desaga, of Heidelberg.

<sup>7</sup> *Loc. cit.*

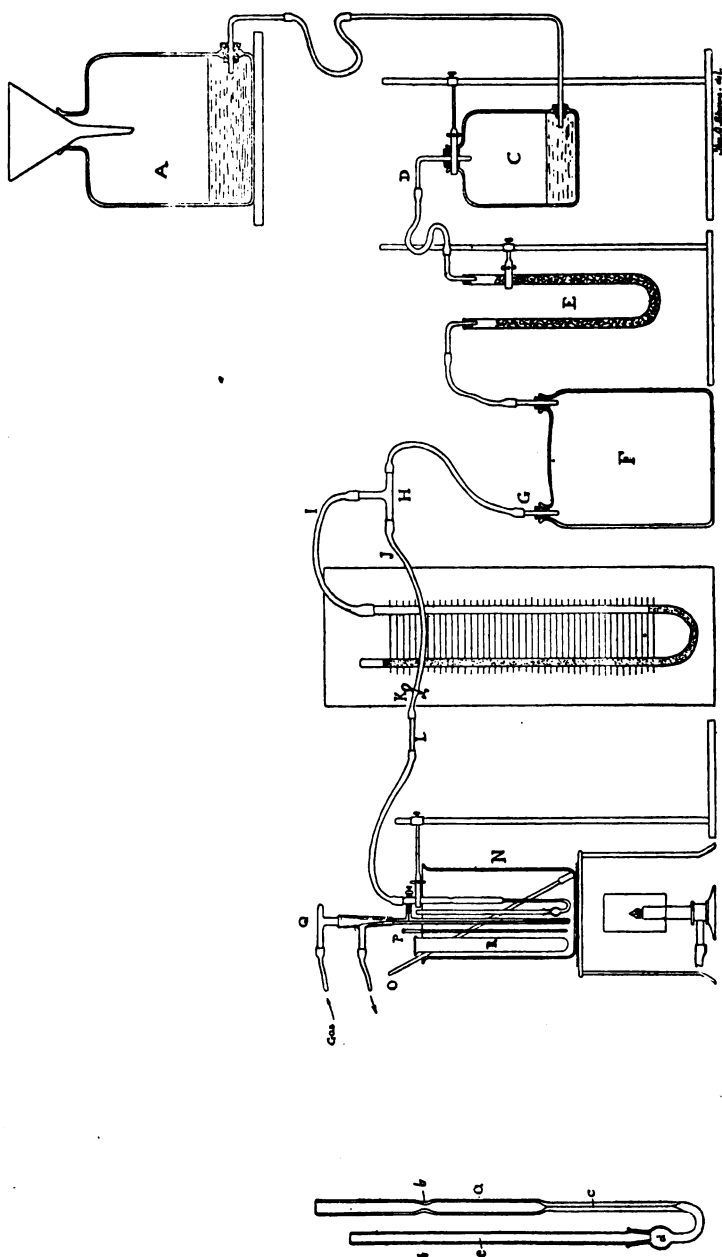


Fig. 1



In the earlier experiments a viscometer made for Hirsch and Beck's<sup>8</sup> apparatus was used; with this the time of flow through the capillary tube was, under the conditions described below, 8.8 seconds for distilled water. This instrument was designed for use with whole blood, the viscosity of which must be observed before it begins to clot. For the present purpose, however, a longer time of observation may be employed, giving greater accuracy; and an instrument was made by Desaga, giving with distilled water a time of flow of 73.1 seconds at 38.5°. This was used to obtain all the curves given in the following pages. The reservoir held 1.44 cubic cm. of fluid; the capillary was 68 mm. long, and its average diameter was .35 mm. A still longer time of flow was not desirable (such, for instance, as half an hour or more, used by earlier workers on the viscosity of fluids, and by Bottazzi<sup>9</sup>), because the fluids observed were undergoing in most cases rapid changes, and it was of advantage to make the time necessary for each observation not too long.

#### MODE OF USING APPARATUS.

The earlier observations were made at a temperature of 38.5 C. It was found later, however, that it was easier to keep the bath constant at 40° C., and as will be seen in many of the later experiments, this was done.

In making an observation, the following steps were taken. The temperature of the bath being constant, the reservoir, d,<sup>10</sup> and the tube, e, were filled with solution to be examined, and e then fitted into its joint at d; fluid fills up the U tube to the lower opening of the capillary; such a quantity of fluid was taken that e was always filled to the same height, namely, to a mark, f, made at the level of the constriction, b, on the other limb of the viscometer; this quantity, in the instrument used, was just under 3 c.c. The viscometer was then put in the bath, the time being recorded, and an interval of five minutes allowed

<sup>8</sup> Loc. cit.

<sup>9</sup> Loc. cit.

<sup>10</sup> See separate drawing of viscometer in Figure 1.

to elapse before taking a reading.<sup>11</sup> Meanwhile, by pulling the glass junction, L, out from the india-rubber tube and sucking, the fluid is drawn through the capillary tube and into the reservoir, and to a point about two centimetres above the constriction, b. L is then again closed, the water in the bath being kept moving by means of the stirrer. The five minutes being up, after a last look at the manometer, the clamp, K, is opened, and the fluid begins to move through the capillary tube. A stop-watch, marking fifths of a second, is held in the hand, and as the meniscus passes the constriction b, the spring is pressed. While the fluid is flowing the water in the bath is stirred. When the reservoir, a, is nearly empty, the observer with one hand takes the watch, and with the other the tube J; it is easy to determine the moment at which air enters the upper end of the capillary tube, and as this occurs the spring of the watch and the tube, J, are simultaneously pressed. The object of pinching J is to cut the pressure off from the viscometer, otherwise the fluid is forced through d and e, and lost. The clamp, K, is now replaced on J, L is opened, and the fluid sucked back into the reservoir. It is especially important when proteid solutions are being used to do this at once, or the solution may dry to some extent on the walls of the capillary tube. The manometer and the thermometer are now again read, and should have remained constant; this being so the time recorded by the watch is noted.

#### THE SOLUTIONS EMPLOYED.

The pepsin solutions were prepared from fresh pigs' stomachs. The separated and minced mucous membrane was washed several times with water to extract as much mucin as possible; it was generally left overnight in water. After straining through muslin, the residue was then extracted with .4 per cent. HCl for twenty-four

<sup>11</sup> It was found with water and with other fluids, whose viscosity at a fixed temperature suffered no change, that their constant time of flow through the capillary tube was reached in less than five minutes. It therefore follows that at the end of this time the temperature of the viscometer and its contents equals that of the bath. It will be seen in the experiments to be described that it was desirable that this interval should be no longer than necessary.

hours, and re-extracted. The second and later extracts contained much less organic material than the first, and were generally not less active.<sup>13</sup> Pepsins III. and V., however, which were employed in many of the experiments, were both first extracts, and were more active than later extracts of the same mucous membrane. The amount of nitrogen in the ferment solution was determined by the Kjeldahl process. One or two drops of chloroform were added to the solutions, and they were kept in a cool place. Under these circumstances Pepsin V. was still strongly active seven weeks after its preparation.

These solutions also naturally contained rennet. It is generally stated that rennet is destroyed by peptic digestion, and with this object the acid extract of the mucous membrane was kept in the incubator at 36° to 40° C. for three days; the neutralised solution did not then cause the clotting of milk: in acid solution, however, it still clotted milk, although when an equal bulk of .4 per cent. HCl was added to milk it remained fluid. Further, if the neutralised solution which did not cause clotting was added in measured quantity to milk, and the same quantity of .4 per cent. HCl added to re-acidify, clotting ensued. This also did not occur in control tubes in which the acid only was added to the milk. A commercial pepsin powder, which was in the laboratory, also possessed the power of coagulating milk.

On looking up this question I found that C. A. Pekelharing<sup>13</sup> has also noted that a pepsin solution which had been digested for five days with .5 per cent. HCl still clotted milk, as did various specimens of commercial pepsins. Hammarsten<sup>14</sup> also quotes the destruction of pepsin by rennet as a frequent but not certain result.

Pekelharing's pepsin, like that used by me, was prepared from the pig's stomach. The observations are explained by the finding, by I. Bang,<sup>15</sup> that the stomach of this animal furnishes a form of rennet (parachymosin) which is much more resistant to digestion than ordinary chymosin.

<sup>13</sup> See Klug. *Loc. cit.*, S. 107. Also Pfüger's Arch., Bd. 60, S. 44, 1895.

<sup>14</sup> Zeitschr. f. Physiol. Chem., Bd. 22, S. 244, 1896.

<sup>15</sup> Lehrbuch. S. 271, 1899.

<sup>16</sup> Deutsche Medicin. Wochenshr, 1899. No. 3.

I kept a portion of the same pepsin extract in the oven for twenty-seven days. It then did not clot milk in acid or neutral solution in four hours at 38° C.; it still retained distinct, though feeble power of digesting fibrin.<sup>16</sup>

Since prolonged digestion in the incubator greatly diminishes the power of the pepsin solution, the undigested rennet-holding extracts were used in the main.

### 3.—EXPERIMENTS.

It may at once be stated that it was found that when a proteid solution is digested by pepsin, its viscosity becomes less; and that this change proceeds at first rapidly, and gradually becomes slower.

This was at first demonstrated with a solution of syntonin, prepared in the following way:—About 500 grams of beef were taken, and the fat and fascia having been cut off, minced, and washed by repeated decantation with water till colourless. To the unfiltered residue water was added to two litres and 16 c.c. of 50 per cent. HCl stirred in, making the percentage of acid in the whole about .4. After twenty-four hours it was filtered through folded and frequently changed filter papers. The solution so obtained was so viscous that it permitted of much dilution without approaching the fluidity of water. It is obvious that if the mixture of proteid and pepsin employed be so weak that its time of flow through the capillary is not far removed from that of water, only a small fall of viscosity can be observed; the employment of an apparatus giving a longer time of flow would, by magnifying the difference, to some extent, overcome this difficulty, but for reasons given above this is not desirable.<sup>17</sup>

It then became necessary to enquire if this phenomenon was entirely due to pepsin acting in the presence of hydrochloric acid, or if such a change occurred with the acid alone, without pepsin, or if, indeed, a proteid in solution underwent any such modification without either pepsin or acid. The reply to the first part of

<sup>16</sup> A comparison was, of course, made with acid of the same strength alone.

<sup>17</sup> See pp. 199—201, above.

the question was easily obtained, for it was found that the syntonin itself diminished in viscosity under the same conditions, the rate of change, however, being much slower than in the presence of pepsin.

With regard to the second part of the question, pure or approximately pure solutions in water were used little in these experiments, on account of the difficulty of getting a sufficiently strong solution. Solutions of various proteids have been examined by Bottazzi,<sup>18</sup> however, with a similar apparatus, giving twenty or thirty times the time of flow of the one used in these experiments, and no such phenomenon is noted by him. Also in figure 5 it can be seen that a solution of plant vitellin in 3 per cent. NaCl undergoes only an exceedingly slight diminution of viscosity at 40° C. This is probably due to the salt, for it is known that salts exert a slow digestive action on proteids.<sup>19</sup> Even if a proteid solution should by itself undergo a change it is so small as to be for the present purpose negligible.

#### 4.—ACTION OF HYDROCHLORIC ACID WITHOUT PEPSIN.

##### ACTION AT BODY TEMPERATURE.

The action of acids on proteids at body temperature has been studied by many workers, and it is known that the same change, usually produced by ferments, can also be more slowly brought about by acids alone. After Meissner had shown that peptone was produced from fibrin by boiling with acid, Wittich,<sup>20</sup> and Wolfhügel,<sup>21</sup> showed that this change takes place at body temperature. Klug<sup>22</sup> found albumoses present with syntonin five minutes after the commencement of the action of weak acid.<sup>23</sup> In Goldschmidt's experiments<sup>24</sup> albumoses were formed from

<sup>18</sup> Loc. cit.

<sup>19</sup> Dastre. *Archiv. de Physiol.*, 1894, pp. 464 and 919; 1895, p. 585, and (with Floresco) p. 701. *Compt. Rend. Acad. Sc.*, 1895, p. 589.

<sup>20</sup> Pflüger's *Arch.*, Bd. 5, S. 468, 1872.

<sup>21</sup> Pflüger's *Arch.*, Bd. 7, S. 188, 1873.

<sup>22</sup> Loc. cit., and Pflüger's *Arch.*, Bd. 60, S. 67.

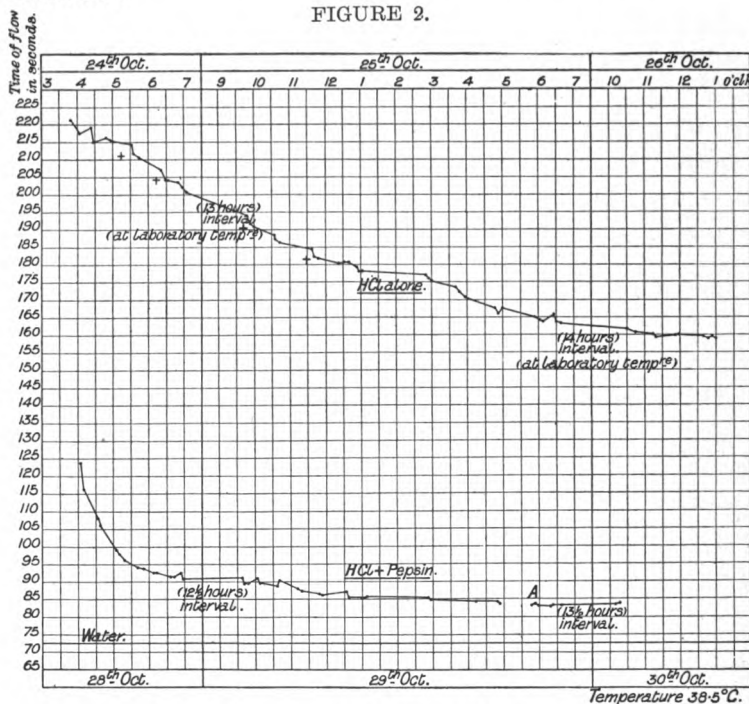
<sup>23</sup> See Sjöqvist. *Skand. Arch. f. Physiol.*, Bd. 5, S. 277, 1895. A review of this subject is here given, with a valuable bibliography.

<sup>24</sup> F. Goldschmidt. *Ueber die Einwirkung von Säuren auf Eiweissstoffe.* Inaug. Diss. Strassburg, 1898.

egg-albumin in one hour at  $18^{\circ}\text{C}$ ., and more abundantly at  $40^{\circ}\text{C}$ . Indeed, albumoses occur so soon that he suggests that an albumose is split off in the formation of acid-albumin. This is opposed by Huppert,<sup>25</sup> who regards this early appearance of albumose as due to the rapid conversion of the first acid-albumin formed.

The importance of acid action is at once brought into prominence when the modification of viscosity in a syntonin solution is observed. The upper curve in Figure 2 was obtained from such a solution.

FIGURE 2.



The vertical row of figures on the left records the time of flow through the viscometer in seconds; horizontally is recorded the time of day at which the observation was made. The readings were taken on three consecutive days, and at night the instrument and the control tube were taken out of the bath. The two vertical lines are divisions between the first and second, and second and third days, and the number of hours the solution remained at the temperature of the laboratory during the night is recorded in brackets on the curves. In this interval the change went on, but much more slowly.

<sup>25</sup> Schütz and Huppert. Loc. cit.

For instance, it can be seen that in the thirteen hours between the 24th and 25th of October the time of flow was reduced by about six seconds, whereas in only one hour in the bath on the next day it fell more than this.

#### INFLUENCE OF MOVEMENT.

In Figure 2 every observation taken is recorded. It was often noticed that when three readings were taken within a short time of one another, a greater diminution in time of flow was found between the first and second than between the second and third; this gives rise to a "step" conformation which can be seen at two or three points on this curve, marked with a cross. This phenomenon is apparently due to the influence of the movement of the fluid in the viscometer, such movement causing an acceleration of the molecular change which the fluid is suffering, and which gives rise to the alteration in viscosity. After this increase in the rate of change the opposite effect occurs, and much less difference is noted between the second and third readings. Indeed, if the curves in Figure 7 (in which pepsin and hydrochloric acid were used) are looked through,<sup>26</sup> it will be seen that in some places there is evidence of a return to a higher viscosity, as if the fluid had somewhat overswung its point of molecular balance. The "step" phenomenon is best observed after the fluid has been at rest for some time.

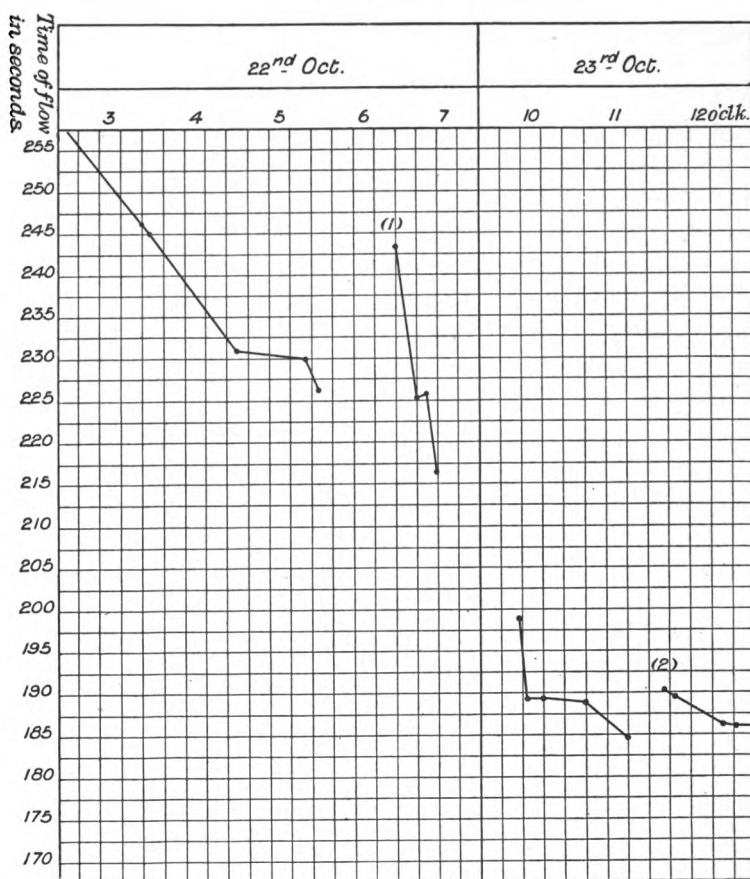
Constant shaking movement of a digesting fluid has been used by various workers to aid the digestion of a powdered proteid, and has been found to increase the quantity of product formed in a given time, presumably by keeping the powder well mixed with the fluid.<sup>27</sup>

In the present experiments there was no question of the distribution of solid particles to be digested, and it becomes important to know if the viscosity of the control fluid in the bath, which remains undisturbed, follows the same course as that in the viscometer, which is moved at intervals. Figure 3 illustrates this point.

<sup>26</sup> Page 218.

<sup>27</sup> See Klug. *Pflüger's Arch.*, Bd. 60, S. 68, and Sjöqvist, loc. cit. In the experiments of Sheridan Lea (*Journ. of Physiol.*, vol. ii., p. 226), and in those of Chittenden and Amerman (*Journ. of Physiol.*, vol. xiv., p. 486, 1893), one object of the movement was to aid dialysis.

FIGURE 3.



Part of some observations made with syntonin III. alone are depicted. The viscosity is rapidly falling, and at 5.32 p.m. the time of flow was 226.8 seconds. The viscometer was now refilled from the solution which had been in the control tube in the water-bath since the commencement of the operations at 10 a.m. Its time of flow was found to be sixteen seconds longer at 6.30 p.m. At the next reading, however, taken in fifteen minutes, it had fallen to 225.4 seconds, showing the step in a marked degree, and in nine minutes more (7 o'clock) the time of flow was further reduced, so that the record would take its place in the falling curve of the original solution. The next day (see 2 in the figure) the viscometer was again emptied and refilled from the control solution. Its time of flow was found to be six seconds longer than that of the previous viscometer solution, and it came more slowly down to the same level. These two differences of sixteen and six seconds respectively are given, because they were the greatest that were ever observed.



From these and many other observations, it was found that the viscosity of the control solution is at any given time a little higher than that of the viscometer solution, and that this is especially so in the earlier stages of an experiment. In the main, however, it follows it, and with a movement such as that of passing through the capillary tube, it rapidly reaches the same level. Obviously the greatness of the movement factor is given by the size of the steps which are found scattered about the curves; they are usually quite small. It may at once be stated that with solutions containing pepsin the correspondence between the control and the viscometer solutions was found to be still closer. Repeatedly the viscometer was filled from the control tube during or at the end of a series of observations, and the viscosity of the control fluid was always found to lie very near to that of the fluid which had been frequently passed through the capillary tube. When some of the control fluid was taken for chemical analysis it was always agitated.

#### THE CHEMICAL CHANGE.

Such a gross physical change as these records show at once suggests that the chemical change which is going on is considerable, and with a view to obtaining information on this point, the amount of coagulable proteid existing in the solution of Figure 2, at the beginning and at the end of the experiment, was ascertained.

By coagulable proteid, here and in the following pages, is meant the proteid which can be removed by neutralising, faintly acidifying with acetic acid, boiling and filtering; that is albumin and acid albumin. The filtrate, with washings, is divided into parts, and in each part the nitrogen estimated by the Kjehdahl method, the mean of the two results, after correcting for increase of volume, gives the amount of nitrogen present as uncoagulable proteid; the total quantity of nitrogen in the solution having been estimated, the amount of nitrogen present as coagulable proteid is found by difference.

The original solution of the upper curve of Figure 2 contained .30 per cent. of nitrogen, of which .18 per cent. was found to be

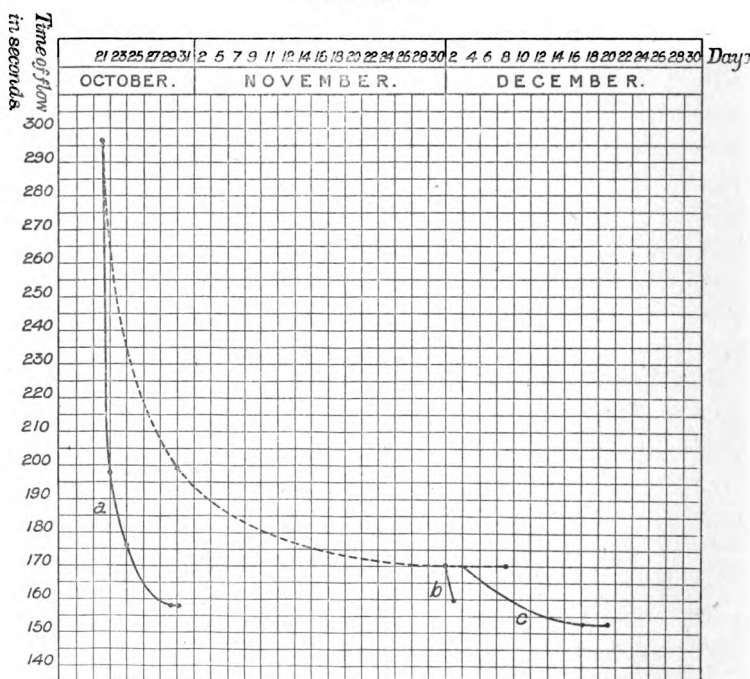
present in the form of coagulable proteid. At the end of the observations this figure was found to be  $\cdot 15$  per cent. It follows that 17 per cent. of the coagulable proteid present in the original solution has been converted into an uncoagulable form.

The amount of free hydrochloric acid present at the beginning and at the end was also determined by titration with caustic soda, using phenol-phthalein as an indicator. No definite difference was found, the figures being  $\cdot 32$  and  $\cdot 33$  per cent. respectively.

#### ACTION OF HYDROCHLORIC ACID AT LOWER TEMPERATURES.

The original solution of syntonin III, kept in the cellar, was at the same time undergoing a change in viscosity, for the time of flow fell in nine days from 297 seconds to 199. In fact, precisely the same process was going on here at a slower rate. The curve which a graphic record gives is at first steep, then less steep, and finally a point is reached at which the change proceeds with infinite slowness.

FIGURE 4.



This figure shows the changes of viscosity which syntonin III. underwent in the course of two months. The time is recorded from left to right in two day periods. The dotted curve represents the alteration in the cold solution in the cellar at about 5° C., each observation being made by putting a sample in the viscometer and taking the time of flow at 40° C. The continuous curves represent the changes in viscosity which occurred in the same fluid at higher temperatures, *a* and *b* being portions taken and kept at 40° C., in the case of *a* for a week and *b* for a day, and *c* is a portion which was put in the incubator at 36° to 37° C. on December 4th and left there for 14 days. The fluid remained sweet throughout the two months.

It is seen that the level at which the process becomes very slow is different for different temperatures ; when at any one temperature it has practically come to an end, if the temperature be raised the solution takes up a new position of molecular equilibrium with a lower viscosity.

#### 5.—ACTION OF PEPSIN AND HYDROCHLORIC ACID.

When pepsin is added to a solution of proteid containing a suitable proportion of hydrochloric acid, the viscosity fall is greatly accelerated. The lower curve of Figure 2 shows graphically this effect. The solution was the same as the one used for the upper curve, except that in place of one part of .4 per cent. hydrochloric acid to nine parts of syntonin III., one part of acid pepsin solution was taken to the same quantity of syntonin. But since the experiment began four days later than the one with hydrochloric acid alone, the initial viscosity of the two solutions cannot be taken as identical, for the viscosity of syntonin III. had in four days itself fallen in the cold ; after this has been taken into account, however, it will be still seen from the curve what a great and rapid fall in the viscosity is caused by the addition of such a proportion of an active pepsin solution as one-tenth of the total volume.

In the case of Figure 2, 14 minutes elapsed between immersion of the mixture in the bath and the recording of the first observation. Since the dilution of syntonin III. with a tenth of its volume of .4 per cent. hydrochloric acid on October 24th reduced the time of flow from about 250 to 220 seconds, that is a little more than one-eighth of the total, it may be calculated that the time of flow of the mixed pepsin and syntonin solution immediately after mixing was not less than 180 seconds ; if this be so the time of flow fell 60 seconds in the first 15 minutes of the action of pepsin.

The process naturally proceeds at first very rapidly because the proportion of unchanged proteid to pepsin is then at its greatest and there is no accumulation of products to hamper the ferment action. Although Chittenden and Amerman<sup>28</sup> found that removing a portion of the albumoses and peptones by dialysis made very little difference to the rapidity of digestion, Kruger<sup>29</sup> has shown that peptic activity sinks with an increased amount of these substances in the solution. It is also notable that the final viscosity of the fluid is by no means far removed from that of water.

At A a sample from the control was put in the viscometer and its time of flow was no greater, as is seen, than that of the fluid which had been constantly moved; the control had been once or twice shaken as samples were removed for chemical analysis.

#### THE CHEMICAL CHANGE.

That the chemical change had been proceeding no less rapidly was shown by the following figures. The original solution contained .31 per cent. of nitrogen (this is .01 per cent. more than the solution of the upper curve of Figure 2, since with the pepsin was added this small amount). It contained .18 per cent. of nitrogen as coagulable proteid. The end solution contained on October 30th, .03 nitrogen present as coagulable proteid. *Hence 83 per cent. of the coagulable proteid was converted, in thirteen hours in the bath, into incoagulable, as against 17 per cent. by the action of hydrochloric acid only, as shown in the upper curve, the solution of which was fifteen hours in the bath.*

#### OBSERVATIONS WITH OTHER PROTEID SOLUTIONS.

It is obvious that to work with a solution such as the syntonin solution, which contains various proteids and is constantly undergoing change itself, is not satisfactory. The presence at the beginning of every experiment of a considerable quantity of uncoagulable nitrogenous compounds alone will retard the rate of conversion of coagulable into noncoagulable forms. It there-

<sup>28</sup> Loc. cit.

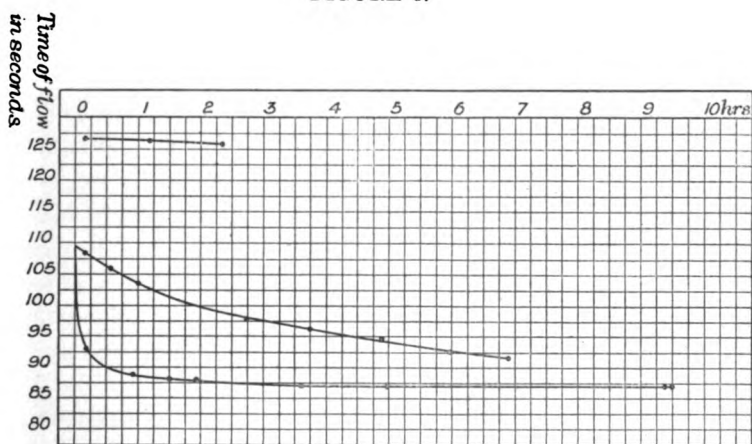
<sup>29</sup> Zeitchr. f. Biol. Bd. 41. S. 467, 1901.

fore became desirable to find some coagulable proteid which could be obtained pure, or fairly pure, in a solution strong enough to give a viscosity sufficiently high for these experiments.

With this object serum-albumin was obtained from horses' plasma by the Hopkins' method and dialysed until no precipitate occurred with barium chloride. The tubes were kept from decomposition by means of chloroform. But attempts to reconcentrate the weak solution thus obtained, in vacuo over pure sulphuric acid, at the laboratory temperature and at 35° C. were unsuccessful. The solution could easily be concentrated down to a certain point, but beyond it was found that apparent further concentration was accompanied by the separation of the albumin in fine particles which remain in suspension, so that after filtering the viscosity was no greater than before. A crystallised sample of edestin, kindly supplied by Professor Kossel, was also treated with warm sodium chloride solution and filtered warm, leaving the insoluble edestin behind. The solution, however, was not viscous enough for the present purpose.

Later on a sample of vegetable vitellin, obtained from Grüber, was found to be soluble enough. Figure 5 is a record of observations made with this substance.

FIGURE 5.



In the upper curve the vitellin is dissolved in warm 3 per cent. salt solution.

The middle curve is from a mixture of 19 parts of a solution of vitellin in 4 per cent. hydrochloric acid (made in the cold), and 1 part of 4 per cent. hydrochloric acid.

The lower curve is from the same acid vitellin solution, but with one part of acid pepsin solution instead of hydrochloric acid.

The figure demonstrates that the same phenomena as are detailed above occur in this purer solution. Unfortunately only a very limited quantity was available, and it must be left to a future communication to describe further experiments with samples of this and other proteids.

Some weeks having elapsed since the first observations of syntonin III. it was found on re-examining it on successive days that its viscosity had become practically constant. One serious objection to its use was thus removed, and as it was available in a quantity sufficient to allow of analyses being made, in spite of the unknown nature of the several ingredients it was employed for further experiments.

#### NATURE OF THE CHEMICAL CHANGE ACCOMPANYING THE VISCOSITY FALL.

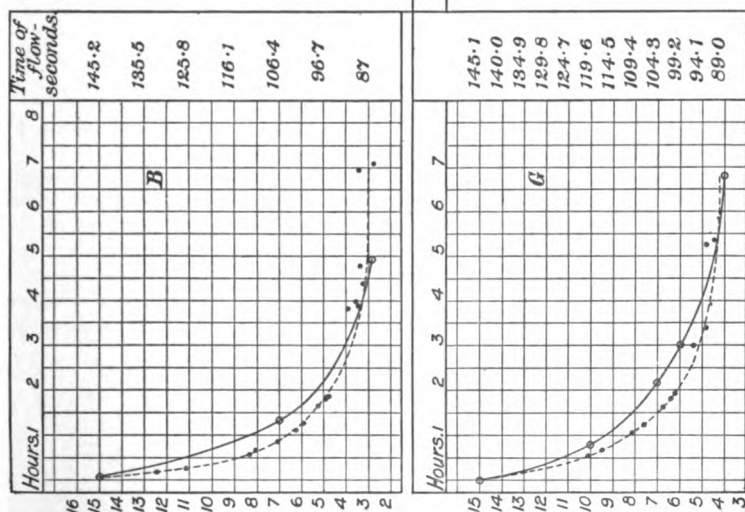
A few considerations as to the nature of the chemical change are here presented; they are, pending further experiments, necessarily only incomplete.

The syntonin solution used and also the acid vitellin solution of Figure 5, presumably consist in the main of bodies of the nature of acid albumin or acid globulin, and it appears that the chemical process underlying the fall of viscosity observed is a transformation of these bodies into simpler substances. There is no direct evidence in the above pages as to whether the change of, for instance, albumin in solution into acid-albumin, would show a similar phenomenon. Indeed, since it is known as mentioned above that when hydrochloric acid acts on albumin solution albumoses can be recognised as soon as acid-albumin it would not be easy to decide whether any fall of viscosity in such a fluid was due to the formation of acid-albumin from albumin only, or to the formation of the lower uncoagulable products, such as albumoses, or to both processes.

It was shown above that in the case of the syntonin solution with and without pepsin, the change of viscosity was accompanied by a decline of the quantity of coagulable proteid present. In a series of experiments made with differing proportions of pepsin, in all cases it was found that the viscosity more and more approached constancy as the quantity of coagulable proteid

diminished; also the viscosity curve approximates to a straight line while there still remains coagulable proteid in the solution. Moreover, if curves representing graphically the diminution of the coagulable proteid are constructed they roughly correspond to the viscosity curves of the same solution.

FIGURE 6.



The dotted curves are the viscosity curves of the solutions B G and E, the figures on the right giving the time of flow in seconds. The continuous depict the diminution of coagulable proteid in the solutions, as ascertained from analyses made, and are constructed by means of the figures on the left, these representing centigrams of nitrogen per cent. present as coagulable proteid.<sup>30</sup>

<sup>30</sup> Similar curves to these curves of the diminution of coagulable proteid have been drawn up by Klug from a series of analyses made by him with the spectrophotometric method (Pflüger's Arch. Bd. 60. S. 57).

It follows that at all events in the main the diminution of viscosity is conditioned by the transformation of the coagulable into incoagulable compounds. As far as the experiments have been at present carried out it has not been determined whether or no the transformation of higher into lower albumoses is accompanied by a similar change, and if so to what extent. It appears, however, that this factor cannot be a great one.

#### EXPERIMENTS WITH ALBUMOSES.

When a sample of proto-albumose which was in the laboratory was dissolved in water and treated with pepsin in acid solution, a precipitate occurred in a few minutes; if a neutralised solution of pepsin were used with a neutral proto-albumose solution no precipitate occurred in six hours and the viscosity did not change. With Witte's peptone and pepsin in acid solution a slight fall of viscosity was followed by a rapid and continuous rise, till five hours<sup>81</sup> afterwards a cloudiness appeared which gradually merged into a precipitate; it is interesting to note that this precipitate was foreshadowed by the rise in viscosity long before an opacity could be detected by the eye. That this precipitate was due to the ferment was shown by the fact that when a control sample of albumose was treated with acid in the same proportion, but without pepsin, the fluid remained clear.

These precipitates at once draw the attention to those obtained by Danilewski and his followers<sup>81</sup> by the action of rennet on albumoses, as that ferment was certainly present in the ferment solution employed. Although, as has been related above, it was easy to obtain a solution of pepsin which would not clot milk in a neutral reaction, a prolonged digestion of the pepsin fluid in the oven was necessary before the coagulative properties were so weakened that a clot was not obtained when the unneutralised ferment was added to milk. At this stage the proteolytic power was also much enfeebled. Such a solution, however, if added to a solution of albumose still gave a precipitate.

<sup>81</sup> Samojloff. Pflüger's Arch. Bd. 85, S. 171.



# 6.—COMPARISON OF CURVES OBTAINED BY TREATING SYNTONIN SOLUTION WITH DIFFERING PROPORTIONS OF PEPSIN.

In order to compare the action of differing amounts of pepsin on the viscosity of a proteid solution a series of digests were made, each containing four parts of syntonin III., and one part containing various proportions of an acid pepsin solution made up to the same volume in every case with .4 per cent. hydrochloric acid. Thus A contained four parts of syntonin and one of pepsin V; B contained four of syntonin and .5 of pepsin and .5 of the hydrochloric acid and so on. A series of solutions was thus made in which the hydrochloric acid proportion was the same in all and the pepsin proportion different. The proteid content was not exactly the same because the pepsin solution contained proteid, and indeed a small quantity of coagulable proteid. The error introduced by this factor, however, is only considerable in the case of the digest A, in which the pepsin solution formed a fifth of the total bulk; the amount of incoagulable nitrogenous compounds added in this case was over 9 per cent. and of coagulable compounds under 1 per cent. of the whole.

In the following table is given the proportion of pepsin in each solution, and the proportion of nitrogen existing at the time of mixture as coagulable and incoagulable substances.

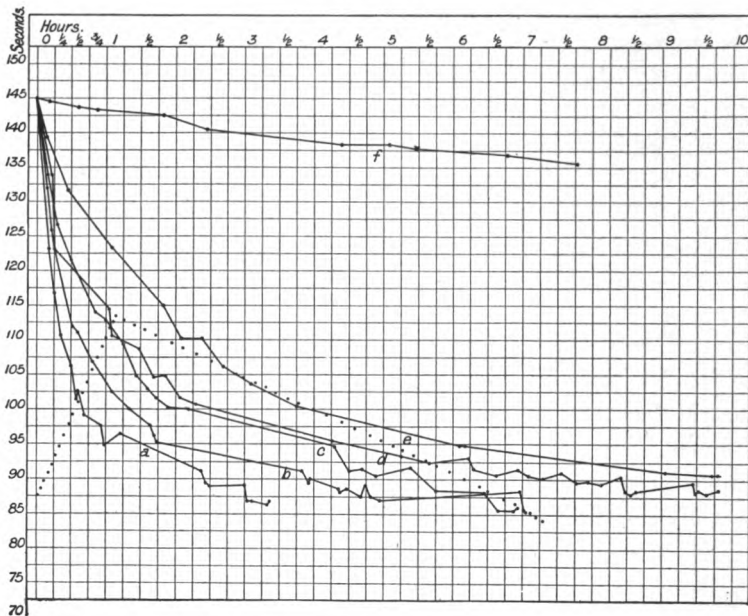
Solution	...	...	...	...	A	B	C	D	E	F	G
Per cent. of pepsin solution	...	...	...	...	20	10	5	4	2.5	.05	5
Per cent. of nitrogen as coagulable compounds	...	...	...	...	.15	.15	.15	.15	.15	.15	.15
Per cent. of nitrogen as incoagulable compounds	...	...	...	...	.14	.12	.12	.12	.12	.11	.12
Total nitrogen per cent.	...	...	...	...	.29	.28	.27	.27	.27	.26	.27

Since the manipulation of these solutions extended over some ten days, it was necessary to be sure that the activity of the pepsin had not altered in this time; to this end the digest G, the last of the series, was made in exactly the same way as C; as its curve corresponded closely to that of C it is seen that the pepsin solution had remained of constant strength.

From this series of digests a series of readings was obtained, the results of which are given graphically in Figure 7. Each

digest was put in the viscometer as soon as mixed, and the time of flow was taken after the interval allowed for the temperature of the viscometer to reach that of the bath had elapsed.

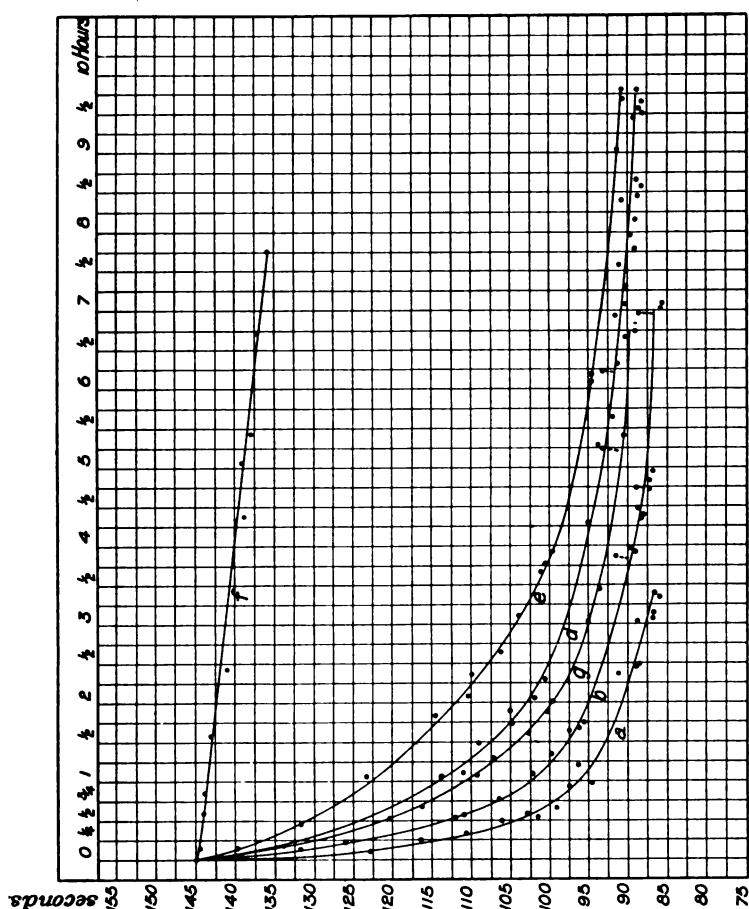
FIGURE 7.



In this figure the numbers on the left hand side represent the time of flow in seconds, and the duration of the experiment is recorded horizontally in hours. The curves were obtained by joining up the dots representing every reading taken. The appearance mentioned above as the "step" phenomenon is seen in several places on the curves.

It will be seen that the initial time of flow of each solution is represented as being 145 seconds, when as a matter of fact the first reading differed in each case, and was not taken for 10 minutes after the solution had been put in the bath. This number was obtained from *F*, which underwent only a very slow change of viscosity, as is shown by its curve, and the first reading of which, taken 10 minutes after immersion in the bath, was 144.6 seconds; hence its initial viscosity was approximately 145 seconds. The time of flow of the other solutions was therefore not less than this, as they all contained a little more proteid than *F*.

If instead of connecting every dot an average curve be drawn, a truer record of the progress of a solution is obtained, as in Figure 8.



In this figure in four cases the dots lie nearer to another curve than their own, and are connected to the latter by a small dotted line. The curve *G* is substituted for *C*.

In the case of each digest when the time of flow became reduced to 100 seconds a sample of 10 c.c. was removed from the previously shaken control and analysed. The following figures give the proportion of nitrogen existing as uncoagulable proteid in each case.

A	B	C	G	D	E
·19	·20	·20	·20	·19	·20

It will be seen that the correspondence is close and it follows, as might be expected, that, in such a series of digests, *each one at a definite viscosity is possessed of the same proportional distribution of coagulable and uncoagulable proteid.*

Hence the method affords a means of recognising in such a series the time at which each one has arrived at a given point of chemical change, as long as the transformation of coagulable into uncoagulable proteid is in progress.

Samples taken at other times of flow also yielded similar results.

Since any given viscosity represents a definite chemical constitution the time taken by any digest to reach a given stage can be read from the curves. It is only necessary to draw a straight line through all the curves at the time of flow corresponding to this stage, and the length of this line from the beginning to where it cuts each curve gives the time in hours for that digest.

A law was enunciated by Schütz<sup>32</sup> to the effect that the rapidity of digestion varied as the root of the mass of pepsin. This result was obtained by measuring polarimetrically the amount of peptone formed. The law held only within certain limits. Borissow<sup>33</sup>, estimating the rapidity of digestion by the quantity of egg-albumin dissolved in unit time came to the same result, and Linossier<sup>34</sup>, using the same method of Mett, confirmed this for pepsin but not for trypsin. Julius Schütz has worked more recently at the same subject with dissolved proteid. He says the root law ceases to hold as soon as half of the coagulable proteid present is digested<sup>35</sup>.

Essentially different results have been obtained by methods in which the digestive solution acted on a solid mass of proteid the surface of which was accessible on all sides. Thus Sjöqvist<sup>36</sup> found, as did Brücke, that the rapidity of the action of pepsin on

<sup>32</sup> Zeitschr. f. Physiol. Chemie. Bd. 9.

<sup>33</sup> Quoted by Samojloff. Arch. de Sc. Biol. Bd. 2, S. 699, 1893. See also Mett. Du Bois Reymond's Arch. 1894, S. 68.

<sup>34</sup> Journ. de Physiol. et Pathol. T. 1, p. 286, 1899.

<sup>35</sup> Zeitschr. f. Physiol. Chemie. Bd. 30, S. 1, 1900.

<sup>36</sup> Loc. cit. S. 358,

solid proteid (in Sjöqvist's experiments in the form of finely divided particles) is proportional to the mass of pepsin.<sup>87</sup>

In my own experiments the subject is approached from another side. Instead of taking unit time and observing the amount of substance formed or dissolved respectively, I have been able to directly measure the time taken to form unit product. If the time taken to reach a given stage could be regarded as the inverse of the rapidity of the digestion the results of this method would be directly comparable with those above quoted: this, however, is only true when the velocity of digestion is constant, although in any given digest the time taken to reach any given stage is of course inversely proportional to the average rapidity of digestion.

The curves show the above-mentioned relationship between pepsin mass and time to a certain extent. Thus in curves A, B, and C, at a time of flow of 87 seconds, if the time (in hours) be squared the following numbers are obtained,

A	B	C
10.9	24.0	42.2

and in these solutions the relative proportions of pepsin are as:—

4	2	1
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This relation of the pepsin mass to the square of the time is given with fair accuracy by that part of the curves in Figure 7 which is included within the two intersecting dotted lines. It obtains tolerably throughout the whole of the curves B and C.

It is obvious, however, that if these curves can be expressed mathematically a much more satisfactory insight into any such proportion as this could be obtained. I have been fortunate enough to be able to obtain the assistance of Dr. Wade, who with great kindness interested himself in the matter, and to whom I am indebted for the following considerations:—

The curves correspond fairly with logarithmic curves of the type

$$\log y = -k (pt^2)^n$$

<sup>87</sup> For the relationships found for other ferments see Duclaux (*Traite de Microbiologie*, 1899, pp. 281 and 590). Also Medvedew (*Pflüger's Archiv. Bd. 65. S. 267*, 1896).

where  $y$  = fraction of diminishable viscosity (which apparently is the same as fraction of residual coagulable proteid).

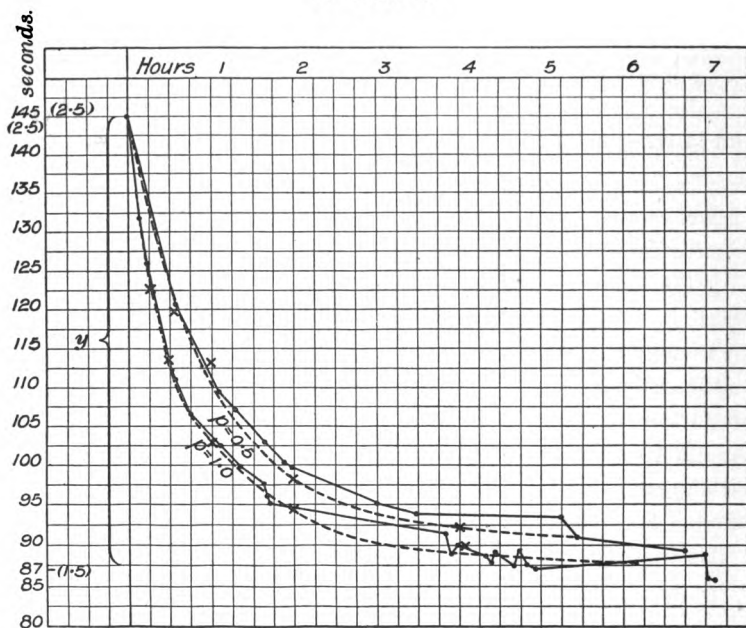
$p$  = relative strength of pepsin.

$t$  = time in hours.

Assigning arbitrary but definite values to the constants  $k$  and  $n$ , and taking the amount of pepsin used in curve  $B$  as unity, the following values of  $y$  were obtained :—

Curve B.			Curve C.		
$p=1.$			$p=\frac{1}{2}.$		
$t$	Calculated.	Found.	$t$	Calculated.	Found.
$\frac{1}{4}$	·62	·62	$\frac{1}{4}$	No observation.	
$\frac{1}{2}$	·46	·43	$\frac{1}{2}$	·55	·57
1	·28	·28	1	·46	·43
2	·12	·15	2	·19	·21
4	·03	·03	4	·07	·10

FIGURE 9.



In this figure are shown two curves (dotted) drawn from this formula, and applied to the actual readings obtained (continuous

line). These curves are only to be regarded as first approximations, and the exact evaluation of the constants must be deferred until the measurements have been repeated on more highly purified proteids. These curves may be found ultimately to merge into the simple logarithmic curves of irreversible chemical action.

Some experiments have been made with other ferments but the consideration of these, as well as of many points suggested by the above enquiry, must be deferred until more work has been done.

### 8.—SUMMARY.

1. The viscosity of a solution of coagulable proteid falls during peptic digestion.

2. A fall also occurs, in the absence of pepsin, through the action of hydrochloric acid, but a much slower one.

3. If the change of viscosity during digestion be expressed by means of a curve, it is seen that it is exceedingly rapid at first, then slower, and finally becomes insignificant; at this time, when the viscosity approaches constancy, most of the coagulable proteid has been converted into an incoagulable form.

4. If samples of the same proteid solution be treated with varying proportions of pepsin, all the solutions when they have reached the same viscosity contain the same proportional distribution of coagulable and uncoagulable proteid. Hence the method offers a means of determining in such a series of digests the time at which the chemical change in each has reached the same point.

5. From the viscosity fall in such a series of digests a series of curves can be obtained, which are capable of mathematical expression, and offer a means of determining the relation between the proportion of the pepsin solution used (or the activity of differing pepsin solutions) and the rapidity of digestion.





# THE GELATIN TREATMENT OF ANEURYSM.

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By E. I. CLAXTON, M.A., B.C.

Being a Thesis for the M.B. Degree, Cambridge.

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## HISTORICAL.

CERTAIN French writers were the first to point out the value of gelatin injections; and, in 1895, M. Dastre et Floresco (1) found that an intravenous injection of sterilized gelatin (1 per cent.), in dogs and rabbits, considerably increased the coagulability of the blood. In 1897, M. Lancereaux et Paulesco (2) employed the remedy in a case of aneurysm of the aorta. Their case was a man, æt. 46, with an enormous aneurysm of the arch of aorta, it existed in the anterior superior part of the thorax as a pulsatile tumour as large as an infant's head (measuring 12 cms. in diameter). The second, third, and fourth right costal cartilages were eroded, and the sufferings of the patient great; the tumour increased in size to 14 cms. in diameter, ecchymotic bosses formed, and the situation became critical. At this stage 55 c.c. of a 1 per cent. solution of gelatin in sodium chloride 1 in 1000 were injected into the subcutaneous tissue of the buttock; the next day the tumour was firmer, diminished daily, and the pain disappeared; but it soon recovered its original size, and the pain returned; a fresh injection of 150 c.c. was administered, and after a dozen of such injections of 150 to 250 c.c., the tumour diminished until it measured only

2 by 1 cms., it was firm, and no longer showed expansile pulsations. In 1898, the patient had maintained a good result for one year, then a fresh pulsating pocket appeared, which was again coagulated by fresh injections. Such, then, is the history of the first case exhibited by the authors at the Paris Academy of Medicine, which they held confirmed their expectations.

## METHODS OF INJECTION AND PREPARATION OF GELATIN ADOPTED.

The methods of sterilizing the gelatin were as follows :—

### METHOD I.

A litre flask was taken and filled with distilled water, sodium chloride being added to make it equivalent to a normal saline solution, 20 grams of gelatin (white) were then added, thus making a 2 per cent. solution, this was warmed in a steam sterilizer for half an hour and the solution then filtered into four sterilized flasks of 250 c.c. each, which were put into the steam sterilizer for twenty minutes at a time on three successive days. The solution just before use was warmed over a spirit lamp to the body temperature. (This method was employed in cases 1, 2, 3, 7).

It was found that the above method was inadequate, and that sterilizing for twenty minutes on successive days in the steam sterilizer was not sufficient to inhibit the growth of the organisms, for on allowing the gelatin so prepared to stand, and making cultivations from it, in five out of eight cases, growth of bacilli were obtained. Hence the following method which most nearly approaches that finally adopted by Lancereaux (3).

### METHOD II.

A 2 per cent. solution of gelatin in normal saline was prepared as before, and sterilized by boiling for twenty minutes in the autoclave at 120° C., this solution was kept for a few days at a medium temperature in order that turbid specimens might be discarded. The gelatin so prepared was found to yield no growth of bacilli on cultivation, nor were its solidifying powers destroyed.

By this method there was found to be less local and general reaction. (Employed in cases 4, 5, 6).

The injections in all cases were made with a carefully sterilized glass syringe and rubber tubing, the needle being introduced into the subcutaneous tissue of the flank, abdomen or axilla, the skin of which had been previously cleansed and carefully washed with an antiseptic lotion (2 per cent. lysol). The injections were made quickly, the time usually occupying fifteen minutes, by this means almost complete freedom from pain was obtained. Never more than 250 c.c. were introduced at one injection, and the number of injections varied from two to three per week. It was found that slight manipulation and massage before withdrawing the needle, hastened absorption of the fluid. A piece of aseptic strapping, or gauze and collodion dressing, was applied to the seat of the puncture after the injection, but a certain amount of leakage took place until complete absorption had occurred.

In all cases the patients were confined to bed during the period of injection, were on a modified diet, and took from five to ten grains of potassium iodide three times a day.

#### TESTS AND ESTIMATION OF GELATIN IN URINE.

*Tests for gelatin.*—Bunge, Sheridan Lea, Halliburton (4)—

Tannic acid gives a precipitate, and yields an opalescence in a solution of 1 in 300,000.

Mercury perchloride yields a precipitate.

Picric acid yields a precipitate.

Millon's reagent yields no precipitate.

Biuret reaction ( $\text{Cu SO}_4$ , and  $\text{KHO}$ ) violet coloration.

Gelatinising power of a solution containing 1 per cent. and upwards.

*Tests in urine.*—Tannic acid and mercury perchloride give a heavy precipitate in any urine. No reaction could be obtained with Millon's reagent and Biuret test in the urine at varying periods after injection.

Picric acid was the only reliable reagent obtained, it gives a slight cloudiness with many *normal urines* and of course always in cases of albuminuria.

By making experimentally standard solutions of gelatin, it was found to give a slight cloudiness with a solution of 1 in 100,000; the most delicate reaction is obtained by pouring a saturated solution of picric acid carefully on top of the urine by means of a pipette, and a resulting cloud is formed between the two liquids. Urine specimens were then taken every hour or two hours for twelve to twenty-four hours after the injection, and on testing by the above means with picric acid solution, a slight cloud was usually obtained on an average two hours after each injection. The best reaction was always obtained in specimens from four to six hours after injection, amounting often to a ring one-eighth of an inch deep; subsequent specimens showed only slight cloudiness, which, however, continued for twelve to twenty-four hours. Attempts to isolate this precipitate and identify it as gelatin were not successful, as it only partially re-dissolved in hot water, and the resulting solution gave a very feeble reaction to picric acid and tannic, and would not gelatinise. The latter test failed probably because of the strength of the solution being less than 1 per cent.

*Methods of estimation.*—

METHOD I.

On comparing the precipitate in urine with the precipitate of standard solutions, the best reaction (one-eighth of an inch ring) obtained three to four hours after injection corresponded to standard solutions of 1 in 5,000 to 1 in 20,000, later specimens (showing only a trace) corresponded to standard solutions of 1 in 50,000 to 1 in 100,000. Closer estimations than these were not possible, as there is not sufficient difference in the precipitates of the solutions of varying strengths, so that by this method a correct quantitative estimation could not be made.

METHOD II.

Other attempts at estimation by precipitation, filtering, drying and weighing were also unreliable because of the infinitesimal amounts, *e.g.*, in estimating a precipitate of 50 c.c. of a solution of different strengths, 1 in 10,000 to 1 in 100,000, the differences in weight were so small as not to be beyond experimental error of the weight of the filter paper.

## METHOD III.

Attempts to estimate gelatin by the amount of nitrogen in the preparation were cut short by cessation of treatment.

Accepting the precipitate with picric acid as gelatin or something very closely allied to it, it does not appear at all possible that any considerable amount of the gelatin injected, is excreted as such; for, according to previous estimate by comparison (Method I.), if one were to suppose that the urine contained 1 in 5,000 parts for twenty-four hours (which estimate is far beyond what was actually found) and taking the total quantity of urine in twenty-four hours as 1,500 c.c., the total amount of gelatin so excreted would only amount to .3 gram after an injection of 200 c.c. of a 2 per cent. solution (= 4 grams), that is only about one-thirteenth is excreted as such.

[The above results were obtained from the urines of cases 1, 2, and 3].

## COAGULATION TIME.

Taken by means of Wright's tubes (four in number) at the temperature of the air of the ward (65° F.).

Varying results were at first obtained; these were found to be due to the differences in composition of the blood while flowing from an ordinary prick in the tissues, possibly to an accumulation of leucocytes at the seat, for on taking blood into the tubes at intervals of a quarter of a minute, the last tube invariably coagulated in quicker time than the others.

It was, therefore, found necessary to puncture a vein, usually on dorsum of hand, so as to get a free flow. Estimating the time in this way before injection and at different intervals of three, six, eight, twelve, and twenty-four hours afterwards, there was found to be *no* appreciable difference.

Coagulation time in case 1 was 3 to 3½ minutes.

" " case 3 " 3½ to 3½ "

NOTE.—In a discussion at the Paris Academy of Medicine in Nov. 1898 (29th Nov., T., xxxv., p. 586, Acad. de Med.).

Laborde held that the estimation of coagulation time of the blood presented the greatest difficulties, partly from faulty

technique, and partly owing to its varying from minute to minute without regularity.

Hayem (*ibidem*) also stated that his results had been so variable and contradictory that he was forced to lay them aside.

### REACTION TO THE GELATIN.

This heading may be divided into two groups : —

#### I. Local.

#### II. General.

In the first is included the pain and discomfort experienced at the seat of injection, while the second deals entirely with the resulting temperature and after-effects of the injection.

#### I. LOCAL REACTION.

Out of fifty-four injections administered in the seven cases quoted, in ten only did pain and tenderness occur, and in two of these abscess formation resulted.

Whether this is due to specific action of the gelatin itself, or is only the result of the stretching of the skin and subcutaneous tissues by the volume of fluid, remains yet to be proved ; but in none of these cases did the local reaction reach such an intense pitch as that quoted by Moynihan (5) in a case (where he gave seven injections and the pain was so intense, and of such a burning character, that gas had to be administered by request for the last five injections). Some writers (Fletcher (6) ) mention the occurrence of hard fibrous nodules over the seat of injection, which disappeared in two to three weeks, nothing of this kind was noted in the seven cases under consideration.

#### II. GENERAL REACTION.

Consists in a rise of temperature a few hours after the injection, sometimes accompanied by a rigor, vomiting, and headache. With reference to the rigors, they only occurred twice in the fifty-four injections, both in case 1 after the first and third injections, and were associated with vomiting.

Headache was variable and was noticed in case 4 once in conjunction with a slight rise of temperature after the second

injection, and again associated with pain and tenderness over the seat of injection. Of these four phenomena, grouped under the heading of general reaction, a rise of temperature seemed to be the most constant, and occurred after thirty-two injections out of a total of fifty-four. In all cases the temperature was taken four-hourly, and the rise took place from two to four hours after injection. Whether the temperature is due to the difficulty experienced in thoroughly sterilizing the gelatin, and consequently bactericidal in origin, or to the action of the gelatin itself, is doubtful; but it is noticeable that cases 4, 5 and 6, which were treated by method of preparation No. II. showed far less general reaction and less rise of temperature than cases 1, 2, 3. No steps were taken to combat this reaction.

NOTE.—Professor Stoicesco (7) recommended and employed 1 gram doses of quinine sulphate before the injections, in order to combat the general reaction and rise of temperature, but apparently without any constant results.

The strength of the solution of gelatin appears to bear no definite relation to the rise of temperature or reaction in general. Before leaving this heading it would be as well to mention some of the chief dangers connected with injection, *i.e.*, the formation of abscesses and death from tetanus, symptoms which occurred in cases 2 and 3; these will be referred to later in detail.

TABLE OF LOCAL REACTION.

No. of Case.	No. of Injection.	Form of local reaction.
1	1st	Pain and tenderness.
2	7th	Pain (I.M.H. administered.)
	8th	Redness, œdema, abscess formation.
3	1st	Tenderness.
	5th	Redness, œdema, abscess formation.

TABLE OF LOCAL REACTION—continued.

No. of Case.	No. of Injection.	Form of local reaction.
4	3rd	Soreness, discomfort.
5		None.
6	1st	Tenderness.
	9th	Tenderness.
	10th	Pain.
7	1st	Pain (refusal of further treatment).

## PATHOLOGY.

It has long been observed that Nature sometimes causes consolidation of an aneurysmal sac; and as the blood has a tendency to coagulate in contact with a vessel whose walls are not perfectly smooth, and whose stream is slowed, and seeing that in most aneurysms the sac is lined by fibrin, it is easy to comprehend that if the coagulability of the blood were increased, the deposit of fibrin would be more rapid and complete, and perhaps sufficient to cause obliteration of the sac. Such was the reasoning which led to the employment of the new remedy gelatin, but the idea is an old one, and various substances were at one time and another employed and justly abandoned.

*Pathological Basis. Experimental Evidence —*

1. Dastre and Floresco (*Archiv. de Physiologie*, Avril, 1896, No. 2, p. 410), have shown experimentally that the coagulation of the blood was considerably increased by intravenous injection of 5 per cent. solution of gelatin in dog and rabbit.



2. Lancereaux et Paulesco (*Acad. de Méd.*, 8 Nov., 1898, T. 40, p. 353) conducted a series of experiments on rabbits, and found rapidity of coagulation of the blood was increased by injecting 100 c.c. of a 2 per cent. gelatin solution into the animal's peritoneal cavity.

3. Boinet (*Archiv. provinc. de Méd.*, 1 Avril, 1899, No. 4, pp. 256 and 259) performed a series of experiments on rabbits, killing and examining them after a subcutaneous injection of gelatin and methylene blue, and found no trace of either; he also made three further experiments, injecting gelatin into the peritoneum, the cavity of which was washed out along with the intestines, and chemically examined, showed almost complete absorption twenty-four hours after the last injection. He found the coagulation time of the blood accelerated in acid gelatin, and not with the neutral after an injection.

4. Geraldini (*Gazz. d. Osped. e. d. Clin.*, 28 Jan., 1900, No. 12, pp. 113 and 115) performed similar experiments with the dog, using injections of gelatin and methylene blue subcutaneously a microscopical examination of the tissues was made after death, and no gelatin found. When performed intraperitoneally, a slight colouration of methylene blue appeared in the urine, and a trace of albumen, and only the slightest trace of gelatin was found in a mass of fibrous tissue adherent to intestines. He found that the coagulation of blood was accelerated after the injection. The author remarks that the trace of albumen points to absorption of the gelatin and partial excretion by the kidneys.

5. Gaglio (*Riforma Medica*, 25-26, Juillet, 1900, Nos. 171-172) states that gelatin injected intravenously takes a very long time to be absorbed, because of its colloidal state; and he thinks the same reason would apply to subcutaneous injection; absorption takes place by the lymphatics.

From these quoted experiments it seems an undoubted fact that gelatin is absorbed, and probably by the lymphatics; if so, in what form is it absorbed? As gelatin or an allied body? In answer to this question it might be pointed out that in section (3) on tests and estimation of gelatin in urine, a body (either gelatin or very closely allied to it) is constantly precipitated by picric

acid after the injection and is excreted by the urine to the amount of one-thirteenth of weight of gelatin injected. There are also two other references to elimination of gelatin in the urine.

Munk and Lewandowsky (Archiv. f. Anat. u. Physiol., 1899, Suppl. Bd. S. 73) showed that the gelatin is partially eliminated in the urine; the rabbit to whom they gave an intravenous injection of 1 gram, 76 of gelatin per kilogram, eliminated 14·8 per cent., retained 85·2 per cent.

Lewandowsky (Berlin Klinisch. Wochenschrift, 3 April, 1900, No. 18, p. 394) thought he had established in man the elimination of the gelatin by the urine, after two injections of 1 gram gelatin, there was in the urine a persistent trace of gelatin, recognisable by a precipitation which it gave on addition of tannin or by heating with acetic acid after saturation with NaCl.

If it is not absorbed as gelatin, or closely allied body, does it undergo peptonization, as is held by Laborde (8)? or, if neither of these two hold good, to what then are its supposed properties due? Why should not its coagulative and hæmostatic properties be due to a common factor? calcium, as Zibell (9) suggests in answering the latter question: he analysed four specimens of gelatin and found that the most constant constituent is calcium, the average amount present being 0·6 per cent. In a 100 c.c. of 5 per cent. solution this would correspond to 0·03 gram of Ca., a not inconsiderable dose, particularly when it is considered that in this form it is very soluble, and therefore very absorbable. The action of a Ca. salts in hastening coagulation is well known, and in the absence of more definite knowledge, it seems highly probable that the gelatin owes its hæmostatic properties, when given subcutaneously, to the calcium it contains.

On the other hand, it seems that the experiments on the coagulation action of the injections on the blood are not convincing partly from the difficulty of technique and partly from the variability of the coagulation times, from minute to minute without regularity or known cause. (*Vide* Laborde et Hayem, Acad. de Méd., Nov. 1898, vol. 34, p. 586).

It seems most necessary to know, if possible, the method of elimination, in case an accumulation in the blood of the coagulative

material might cause progressive coagulability of the blood, and finally massive coagulation.

Incidentally, before leaving the pathology of the subject, we might enquire into the effect of the volume of fluid injected on the circulation and aneurysmal sac itself. The introduction of a large quantity of artificial serum into the circulation gives rise to increased blood-pressure, and as the gelatin is contained in a normal saline solution and about 250 c.c. are injected, what effect would this have on the blood-pressure?

Now, Dastre et Floresco (1) have shown that the pressure is increased by the gelatin itself and its effects are superadded to those of the artificial serum.

Grenet et Piquand (10) have noticed in their own personal observations following an injection, a rapid increase in volume of aneurysm, and an accentuation of the signs of compression; phenomena which they think point rather to increased blood-pressure. Somewhat similar results were at times noted after third, fourth, seventh, and eighth injections in case 1, when the aneurysmal sac seemed tenser and the pulsation harder.

But, on the other hand, Barié made observations with the syphgmomanometer of Potain on arterial pressure before and after injection without ever finding increased pressure. To obviate this difficulty of injecting a large quantity of gelatin solution, and to avoid if possible the production of a vicious circle, De Castro y Latorre (11) uses stronger solutions, 3 grams in 40 c.c. distilled water (no salt), and injects 1 to 4 c.c. in the proximity of aneurysmal sac in the course of the lymphatics.

The pathology of the gelatin treatment may be summed up as follows:—A means to imitate the natural process of clot-formation in an aneurysmal sac; a process no better than the treatment by trying an introduction of wire, galvano-puncture, acetate of lead, alum, tannin, perchloride of iron, calcium chloride, and other coagulants. Does this constitute a cure in the true sense of the word? Is not the general atheromatous state of the aorta overlooked? And even if one dilated pouch is filled with a laminated clot, are there not many incipient aneurysmal

pouches, such as were pointed out by Professor Coats, ready to give way and produce another actual aneurysm?

The conclusions drawn are that the gelatin treatment is not based on the true pathology of the disease, and must therefore be essentially inadequate.

NOTE.—Coats and Auld, on Path. of Aneurysm and Enderitis Deformans, Journ. Path., 1897, p. 105. “In three hundred consecutive post-mortem examinations carefully recorded with special view to this point the “beginnings of aneurysms” in the shape of slight depressions or pouchings of the coats, were found in eight cases, three of which were cases of sacculated aneurysm.

#### DISCUSSION OF CASES, CHOICE, DANGERS, &c.

It is quite impossible to judge the value of a method of treatment without a considerable number of cases, and as only seven cases are here recorded, deductions from them alone would be fallacious. So all I here propose to do is to follow the plan adopted by Grenet et Piquand (12) who analysed one case from their own personal observation together with 100 others from literature, and see how far the seven cases referred to fall into their groups of classification:—

*First group* (11 cases).

Observations without value with regard to result of treatment.

*Second group* (5 cases).—Grave sequels, ? due to treatment.

*Third group* (25 cases).—No apparent benefit.

*Fourth group* (49 cases).—Temporary or permanent improvement. (Grenet et Piquand, p. 651).

In the *first group* case 7 appears.

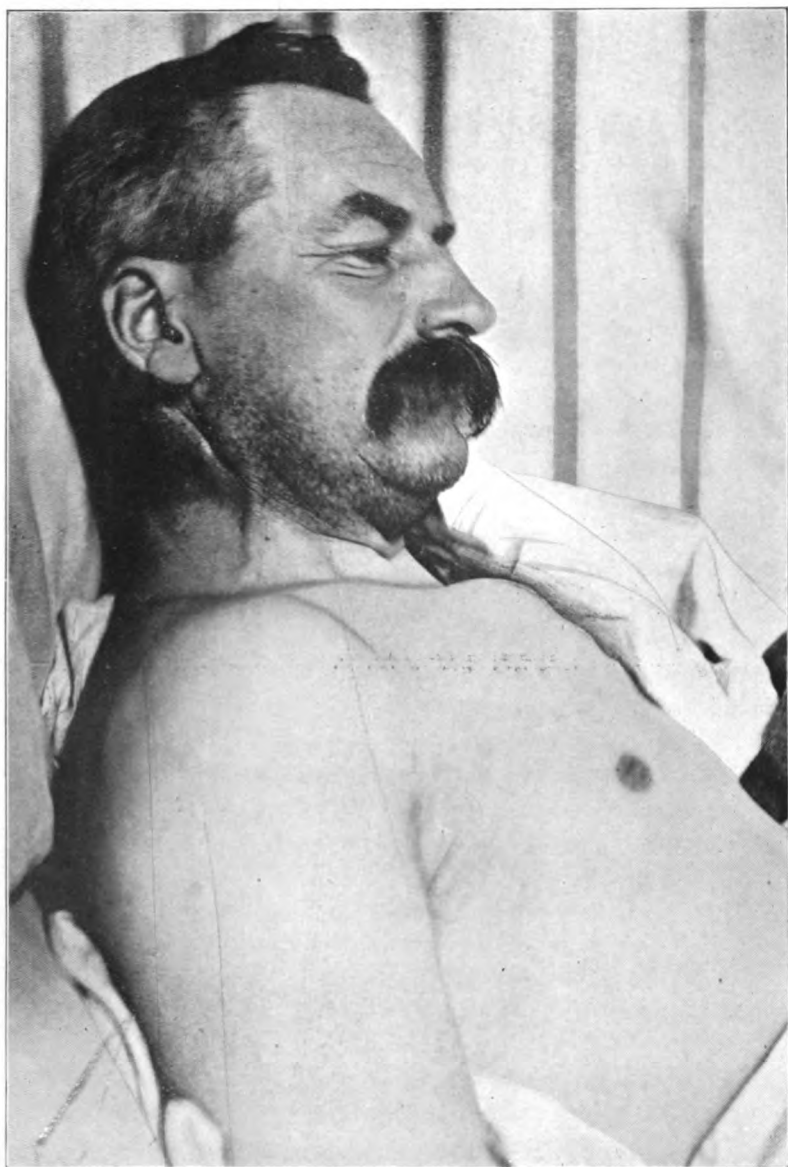
Case 7. Only had one injection (3 ounces of 2 per cent. solution), and refused further treatment because of the pain of the injection. Result nil.

In the *second group* cases 2 and 3 appear. Both died from tetanic symptoms.

Case 2. No visible tumour, paralysis of left vocal cord, and dyspnoic attacks, had eight injections of 250 c.c. of 2 per cent.

*The Gelatin Treatment of Aneurysm.*

CASE No. 5.



[Notice the hour-glass-shaped tumour over 1, 2, 3 spaces.]



to 5 per cent. solution. Some amelioration of subjective symptoms took place. After the eighth injection seat of needle puncture became red and swollen, next day temperature rose to 100°, later swelling and œdema, and finally abscess formation. Five days after last injection trismus set in, and was followed nine hours after by general tetanic convulsions; abscess opened, and foul pus evacuated; death. Bacteriological report of pus and gelatin used for injection. No tetanus bacilli could be found, but a large quantity of short motile bacilli.

Case 3. Abdominal aneurysm of coliac axis; had five injections of 250 c.c. of 2 per cent. to 5 per cent. solution; after the five injections temperature rose to 100·2° next day, seat of needle puncture red and tender; five days after last injection (one hour later than case 2), trismus set in, and general spasms one hour later; abscess formation, abscess opened, foul pus evacuated, death from tetanic convulsions.

In the *third group* cases 4, 5 and 6 appear.

Case 4. Saccular aneurysm of arch, had seven injections of a 2 per cent. solution, 250 c.c. at a time; she had pressure on superior vena cava and œdema of face and arm, and right pleuritic effusion, for which aspiration was twice performed. Venesection twice, ten days after the second paracentesis thoracis, patient gradually failed, became comatose and died. Result: no apparent amelioration after any injection.

Case 5. Aneurysm of ascending aorta; had eight injections of 2 per cent. to 4 per cent. solution of gelatin, 250 c.c. at a time with no apparent result. During the treatment pulmonary symptoms gradually increased and eventually carried off the patient three months after the commencement of treatment. Result: No apparent amelioration after an injection.

Case 6. Came up specially for the treatment by gelatin, was under treatment from October to December, 1900, and during that time had seventeen injections of a 2 per cent. to 4 per cent. solution; was discharged, as his condition remained unchanged. According to the patient's statement he derived much benefit; died in February of bronchitis. Result: no apparent benefit, some amelioration of subjective symptoms.

In the *fourth group* case 1 appears.

Case 1. Saccular aneurysm situated in first and second right spaces, measuring 2 inches by  $2\frac{1}{2}$  inches, had eight injections, the first followed by rigor, temperature and vomiting, reduction of tumour in forty-eight hours; after second injection further reduction, measurement one inch and a quarter in diameter, and barely prominent. Patient gets up and walks upstairs without discomfort. After third injection size of tumour again diminished perceptibly, pulsation more distant; further diminution in size after last two injections (*vide* photographs). Patient discharged three months after, much relieved.

Later—Patient has been under observation from time to time, and in October, 1901, when seen, had some recurrence of pain in the chest but no increase in the size of tumour.

May, 1902. Patient has been lost sight of up to the present.

Result.—Temporary amelioration.

Therefore out of seven cases which were all treated similarly, *i.e.*, with Pot. Iodide, complete rest in bed and a varying number of gelatin injections:—

One case, observations were without value.

Two died from grave sequels due to the treatment.

Three derived no apparent benefit, and one case has, so far, derived temporary benefit.

Results, which are very similar to those obtained by Professor Gouloubinine (13) who, out of eight cases, had one that derived some temporary benefit, four died, and three were lost sight of.

*On choice.—Indications:—*

1. M. Lancereaux (2 and 3) points out the efficacy of the injection in *sacculated aneurysms* only, which is not likely to be so useful in single fusiform dilatations, because coagulation takes place less readily, owing to absence of slowing of the blood-stream, and the danger of obliterating the whole vessel.

The seven cases here quoted all belong to the above category.

2. M. Regett (14) points out the importance of the fact of date of affection, relatively recent aneurysmal formations being more favourable to coagulation.



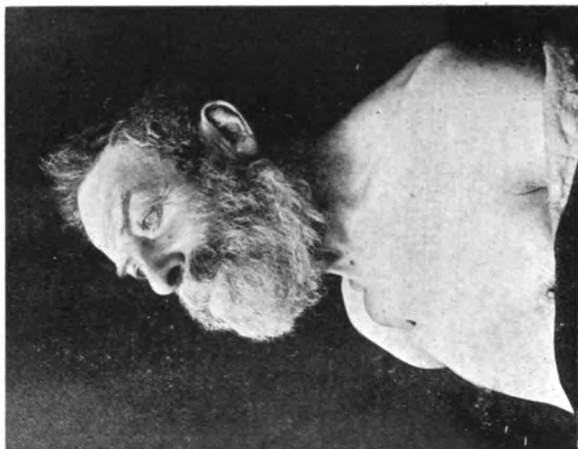
*The Gelatin Treatment of Aneurysm.*

CASE No. 1.

Taken before commencement  
of treatment by Gelatin.

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May 16th, 1901.





*The Gelatin Treatment of Aneurysm.*

CASE No. 1.

Taken after the 3rd Injection.  
June 24th, 1901.

—  
[Showing marked diminution  
in size of tumour.]





In the seven cases, the periods vary between three weeks, and three and a half years, but there is some difficulty experienced owing to the patients dating the commencement of their trouble from the pain, in some cases not having noticed the tumour; in cases 1 and 3 only, was the latter noted, and the dates are six months, and three weeks respectively.

*Contra indications.*—The danger of injection in a very thin-walled aneurysmal sac, leading to increased blood-pressure, by means of the large volume of serum and gelatin injected, has already been mentioned in the section on Pathology.

*Dangers of the treatment.*—There is really but one great danger in the treatment, and that is *sepsis*. How far, then, is it possible to make the gelatin sterile without losing its imputed properties of promoting coagulation of the blood? There have been several recorded cases of death from tetanic symptoms:—

1. Kuhn (Wein Med. Woch., Jan. 18, p. 138). Med. Review, p. 240, Apr. 1902, where tetanus developed in a hæmophilic boy after removal of adenoid vegetations, an injection of gelatin being administered as a hæmostatic. After death tetanus bacilli were found and proved conclusively by experiments on rabbits to have been injected through needle track.

2. Kuhn. Tetanus nach gelatine-injection. (Münich Med. Woch, 1901, s. 1923).

3. Maguire (Soc. of London Med. Trans., 1901, vol. 24, pp. 50 and 51) records one death from tetanic symptoms four days after injection though there was careful asepsis; the only explanation was that the patient's room was practically in a garden, and it is well known that garden soil is the habitat of the tetanus bacillus.

4. Freudweiler (Centralbt. für Klin. July 7th, 1900) fatal case from tetanus.

5. *Lancet*, vol. 2, 1901, p. 610. *Brit. Med. Jour.*, 1901, vol. 11, p. 638, references to the death of two cases by tetanus (which are cases 2 and 3 here recorded).

Though bacteriological examination, both of the pus from abscesses and the gelatin solution used, was negative with regard to the tetanus bacillus, there seems no doubt whatever that the

symptoms were clinically those of tetanus, coming on five days after injection.

The following abstract from the *Medical Review*, April, 1902, pp. 240-241, of the work done on "Presence of tetanus spores in commercial gelatin," by Levy and Bruns (*Deutsche Med. Woch.*, Feb. 20, p. 130) is interesting, and is here inserted verbally, showing the difficulties of complete sterilization without destroying the inherent properties of it.

"Levy and Bruns have examined six samples of commercial gelatin and found tetanus spores in four. From two to three grams of gelatin were dissolved in 100 c.c. of broth, and the solution was placed in the incubator at 37° for eight to ten days. Other spores adhering to the gelatin, the tetanus spores developed in the lower layers of the culture into bacilli, and secreted the tetanus toxin. The culture was then filtered and 3 to 4 c.c. of filtrate were injected in mice, which died within two or three days of typical tetanus. In one case, a smaller dose (2 c.c.) produced chronic tetanus of the posterior limb, into which the injection was made. Four to five cubic cms. of the filtrate were fatal to the guinea-pig. In the dirty greyish sediment which formed in the cultures, typical drum stick bacilli with terminal spores were found, and the direct injection of this sediment into animals usually produced tetanus. The method, however, is not advisable, since other spores and bacilli present, especially of malignant oedema, may kill the animal before tetanus appears. Some of the spores or the spore forming organisms present in gelatin are very resistant, and heating to 80° C. for one hour was insufficient to obtain pure cultures of the tetanus bacillus. The question whether it is possible to sterilize gelatin sufficiently for hypodermic injection is at present unsettled. It is usually stated that a jet of steam at 100° C. will kill the spores of tetanus in eight minutes; in the writer's experience this is insufficient. The spores of any organism vary in resistance according to the source, and it is possible that this liability is true of some specimens of tetanus bacilli. Another unsettled question is whether sufficient prolonged heating does not destroy the value of gelatin as a hæmostatic. If, as Zibell maintains, (*Münich Med. Woch.*, No. 42, 1901) the

active principle is the calcium chloride contained in gelatin in a soluble form, prolonged heating to 100° or more would not destroy it. If due to power to 'set,' prolonged heating would be prejudicial."

Before leaving the subject, it would be as well to mention other recorded dangers, such as coagulation *en masse*, and embolism by gelatin.

Kingston Fowler (Med. Soc. of London, December 10th, 1900, in *Lancet*, 1900, vol. 11, p. 1732), records a case of a female, æt. 48, with an aneurysm of transverse portion of aorta, who, five days after a second injection of 150 c.c. of 2 per cent. solution, was attacked by neuralgic pain, vomited, became restless, collapsed, and finally unconscious; she recovered two days later complaining only of pain down neck and back. The author thought this due to symptoms of cerebral embolism and blocking of internal carotid.

Barth, in Huchard (Bulletin, de l'Acad. de Médec., October 25th, 1898, T. 40, p. 313, et Journ. des Practiciens, 1898, p. 708), records a case of aneurysm of ascending part of aorta, in which, after six injections of 2 per cent. solution, there was much pain at seat of injection, and following day rapid rise of temperature to 40° C., for twenty-four hours, then a fall to normal. A large abscess formed, and death ensued from collapse. Post-mortem coagulation *en masse* of the large arterial trunk at origin (innominate and its branches and left carotid).

Lancereaux criticised the above (Bull. de l'Acad. de Méd. 8th November, 1898), and held the symptoms resembled those of uræmia. Huchard replied (15th November, 1898, p. 418) the urine had been examined, and uræmia did not appear to be the cause.

Laborde (Acad. de Méd., 31st, October, 1898, T. 40, p. 336) thinks the gelatin is not dissolved but remains in suspension, and that the solid particles thus introduced in the circulation might be the starting point of an embolus.

Grenet et Piquand (15) think the embolic view more theoretical than real, and it is difficult to know whether the embolus is due to injection of gelatin or merely dependent on presence of the

aneurysm itself; it is also possible that the injection may render the detachment of aneurysmal clot more frequent. With regard to coagulation *en masse*, the autopsy of Barth appears conclusive enough; but even if the gelatin were directly injected into the vessels would such a dose be sufficient to produce massive coagulation? De Castro y Latorre (11) used stronger solutions, and injected them into the proximity of sac without any dangerous results. It is interesting to note that Groves (16) in referring to Freudweiler's cases of tetanus states that the symptoms were rather uræmic than tetanic, renal symptoms being conspicuous. He supports this on the analogy that convulsions of eclampsia may be due to increased blood coagulation leading to multiple thrombi in the brain, kidney and liver; why, therefore, should not these symptoms be due to capillary thrombi in nervous tissue?

Broadbent (*ibidem*, p. 741), too, inclined to the view that in the two cases, 2 and 3, the oncoming convulsions were rather rapid for tetanus, and also suggests that they may be due to gelatin thrombi in minute vessels of spinal cord affected by atheroma already existing.

#### DISCUSSION ON OBSERVATIONS MADE IN SECTIONS 2 TO 6.

With regard to *methods of injection*, the following at one time and another have been employed.

1. Subcutaneous injection.
2. Intra muscular injection.
3. Rectal injection.
4. Subcutaneous injection in the proximity of the sac.

1. With regard to the subcutaneous injection, nothing further need be said.

2. Lancereaux (3) holds that the intra muscular injection has the advantage of rapidity of injection, absorption by deep lymphatics, and is painless, and is therefore to be preferred to the subcutaneous method.

3. Rectal method.—Racchi (17) suggests an improved method of using gelatin for hæmostatic purposes, ten drachms of warm 2 per cent. gelatin in normal serum is injected per rectum, and is more rapidly absorbed.



Boinet (18) along with subcutaneous injections, used rectal injections of concentrated gelatin, night and morning, in a case of aneurysm, in which death occurred by syncope. Post-mortem, pressure on the pulmonary artery was found.

4. De Castro y Latorre (11) avoids weaker solutions, which, as he considers, produce rapid coagulation in the aneurysm, and uses 1 to 4 c.c. of a concentrated solution injected subcutaneously in close proximity to sac, and in the course of lymphatics. This is quite contrary to the teaching of Lancereaux, who states that the injection should never be made near the aneurysm.

Gaglio (19), after studying the chemiotaxic properties of gelatin, suggests that gelatin injected in the neighbourhood of the sac might be capable of producing retraction of the sac by an irritative local action.

*With regard to apparatus for injection.*

The simplest seems to be the best, as it can be more easily and readily sterilized (hence the use of flask and glass syringe only in the seven recorded cases).

Maguire (20) strongly recommends the use of an apparatus for intra muscular injection, made by Messrs. Squire & Sons, where the principle employed is simply that of an ordinary wash bottle, the liquefied gelatin being forced through the tube by a current of air from a large brass syringe previously sterilized by carbolic acid. (The apparatus is depicted in *Brit. Med. Jour.*, 1901, vol. 2, p. 1414).

Herman Gardner (*Brit. Med. Jour.*, 1901, vol. 2, p. 1274) has invented a rather complicated apparatus to get rid of the imperfection in filtering, which he states is the drawback to perfect sterilization. He devised an apparatus whereby perfect sterilization is obtained, since the injection when filtered may be boiled in it and retained until use.

With regard to the difficulties of perfect sterilization enough has been already said, and it seems still an unsettled question (*vide* Levy and Bruns on Tetanus Spores in Commercial Gelatin.)

*Treatment during injection and after-treatment.*—Dr. Theo. Fitcher (*Ann. Journ. of Med. Sc.*, 1900, p. 603), quoting Lancereaux, states that a proper diet is imperative; strict milk diet

preferred, fruit and leguminous vegetables may be permitted. Iodides and nitrites are indicated and strict confinement to bed necessary.

Fräenkel (Deutsch Med. Presse, 1899, p. 88) (Med. Review, vol. ii., 1899, p. 544) says a special dietary and complete rest in bed help coagulation.

The above rules were followed in the seven cases recorded, more especially in case 1, where a special dietary was ordered.

*Tests and estimation of gelatin.*—With regard to the above complete and accurate estimation was not found possible, and the gelatin, or allied gelatin body is only excreted to the amount of one-thirteenth as such. Two other observers (Munk and Lewandowsky) to whom reference has been already made, have shown that gelatin is partially eliminated in the urine. Nothing further than what has already been mentioned need be said on the question of coagulation time of the blood.

*Reaction.*—Most observers are agreed that there is a local and general reaction, and that it is due to action of the gelatin itself; though Lancereaux (21) states that with properly sterilized gelatin, and due antiseptic precautions, there ought to be no local or general reaction.

Teresi (22) thought the pain during injection due to the volume of gelatin injected and strength of the solution. From the observations on the seven cases, the bactericidal view seems to be the most probable.

*Pathology.*—Very little need further be said on this subject; but just one point might be mentioned with regard to the clot formation. In the autopsies of the four cases here recorded, *laminated clot* was present in three of them, in amounts of—

Case 4.—Two-thirds of total clot.

Case 5.—One-half to three-quarters of total clot.

Case 2.—Clot one inch in thickness.

*while recent clot* was present in three cases.

In amounts of—

Case 2.—One-eighth to a quarter of an inch in thickness.

Case 3.—Very flimsy indeed.

Case 4.—One-third of total clot.

The probability is, that in the majority of cases where laminated clot is present, it was there before treatment began; it is questionable even if it might be increased by treatment, but extremely doubtful whether it could be created by it; estimation of improvement is fallacious, because the tumour under observation is at the best a very small portion of the sac, and there might be coagulation there while the sac was extending in another direction.

### CONCLUSIONS.

That 1. The method is an extremely dangerous one and apt to result in death from tetanus; this is due to the difficulty experienced in the sterilization of the gelatin without destroying its inherent properties.

2. Great difficulty is experienced in estimating (quantitatively and qualitatively) whether gelatin, or a closely allied body, is excreted in the urine.

3. The coagulation of the blood is not appreciably increased by injections, varying results being obtained owing to difficulty of technique.

4. There is a reaction to injection local and general, which is in all probability bactericidal in action.

5. In no case has there been any cure, and at the best, only slight amelioration; expectation of improvement fallacious.

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## LIST OF CASES.

CASE 1.—E. W., æt. 61, male, was admitted May 7th, 1901, under the care of Dr. Perry, for swelling on front of chest. *Personal history*.—Married, always a hard worker, lately employed in excavating, and during life occupied as coal heaver, lime loading, etc., used to drink very heavily. *Previous illnesses*.—Ague at 28, ill six months; influenza ten years ago, winter cough of four or five years, slight hæmoptysis six years ago, no definite specific history. *History of present condition*.—Just after Christmas, 1900, noticed small swelling in front of upper part of chest, hard, not painful. Six months previously patient had pain across chest, especially after exertion, this got worse and since Christmas the swelling has increased. The pain made him feel sick.

*Condition on admission*.—Pulse 72, temperature 98·4°, respiration 24. A healthy looking man with grey hair and blue eyes. There is an oval shaped swelling slightly to the right of upper end of sternum, situated over the cartilages of the first and second ribs, and manubrium sterni; it measured two inches long and two and a half broad; the skin over it is normal; the swelling pulsates strongly synchronously with heart, is soft and expansile, not painful to the touch, a dull note on percussion is obtained over it. There is visible pulsation in the arteries of the neck: the axillary and brachial arteries also pulsate, but more on the right side. No bruit over the swelling but a distinct diastolic shock can be heard; no swelling of arm or tracheal tugging present.

*Circulatory system*.—*Heart*; impulse seen and felt in the sixth space just external to nipple line, no cardiac dullness, sounds loud, no bruit present, *Pulses*: equal, good tension, rate 72. *Special senses*: eyes react to light and accommodation, pupils equal, knee-jerks present. *Urine*: acid, clear, mucus present, sp. gr. 10·28, urea 2·4 per cent., no albumen. Other symptoms normal.

*Progress and treatment*.—May 7th, Pot. Iod. 5 grains, t.d.s.

May 9th. *Diet*: Breakfast, bread and butter and a small cup of tea. Lunch, beef tea and toast. Dinner, meat 4ozs., small amount of vegetables, a quarter of a pint of fluids. Tea, small cup with bread and butter. Supper, soup or beef tea and toast. Cui adde mist., five grains of Pot. Iod.

May 16th. Condition the same, some pain in chest. Pot. Iod. increased to 15 grains.

May 21st. Iodide rash on chest. Pot. Iod. increased to 30 grains.

May 24th. Iodide rash disappeared, condition otherwise unchanged.

June 8th. *Injection of 250 c.c. of gelatin* (2 per cent.) in the morning followed by rigor two hours after, and a rise of temperature to 101·2°. Vomiting. Reduction of tumour in forty-eight hours. Some pain and tenderness over seat of injection (left side of abdominal wall). Plaster cast taken just before injection.

June 11th, 1.15 p.m. *Injection of gelatin*, 100 c.c. (2 per cent.) Temperature rose to 100°; no rigor. Gelatin found only in first sample of urine, of which six specimens were obtained hourly, viz., 2, 3, 4, 5, and 8 p.m. The first specimen of 40 c.c. gave a precipitate with picric acid, no result in others; a further reduction of tumour.

June 13th, 3.10 p.m. *Injection of gelatin*, 200 c.c. (2 per cent.) into left flank. 7 p.m., rigor and vomiting, temperature 99·8°, at 10 p.m. temperature 102·4°. A precipitate of gelatin found up to 7 p.m. in urine, specimens being secured half hourly up to 6 p.m., and hourly up to 10 p.m.

July 3rd. Patient's temperature has remained either normal or subnormal. Since the 15th inst. he feels well and has no pain. Tumour extremely small (*vide* photo of 24th June), and feels much firmer. He has been getting up after dinner for a fortnight, and can walk upstairs without discomfort, the superficial prominence of the aneurysm is now barely one and a quarter inches in diameter.

July 4th, 2.30 p.m. *Injection of gelatin*, 180 c.c. (2 per cent.) into right flank, a rise of temperature to 99·8°. Gelatin found in first and second specimens of urine only. Next day size of tumour diminished perceptibly and pulsation feels more distant.

July 11th. Patient skiagraphed to-day; on the screen nothing definite could be made out, but a diffuse obscure mass was seen over the arch of the aorta.

July 15th, 4 p.m. *Injection of gelatin*, 250 c.c. (2 per cent.) into right flank. 10 p.m. temperature rose to 99·2°. Gelatin found in first three specimens of urine only.

July 18th, 4.30 p.m. *Injection of gelatin*, 220 c.c. (2 per cent.) Urine shewed presence of precipitate three hours after, and about half the amount in five to eight hours after injection; eleven hours after no trace. The tumour has not seemed to diminish in size during the last week.

July 27th, 5.30 p.m. *Injection of gelatin*, 200 c.c. (4 per cent.) followed by no rise of temperature, swelling harder and firmer, pulsation more distant.

August 7th. *Injection of gelatin*, 200 c.c. (5 per cent.) followed by no rise of temperature. Gelatin found in urine six hours after.

August 19th. Patient discharged well, walking without discomfort. There has been considerable diminution in the tumour during the last fortnight; pulsation firmer and more distant.

October, 1901. Patient since discharge from the hospital has been doing some heavy road mending work, which has caused recurrence of the pain in the chest, apart from this he seemed comfortable and the tumour had not appreciably increased in size. Since this date patient has been under observation from time to time, and has been employed in doing light work; he has been feeling well and there has been no further increase in the aneurysmal sac again.

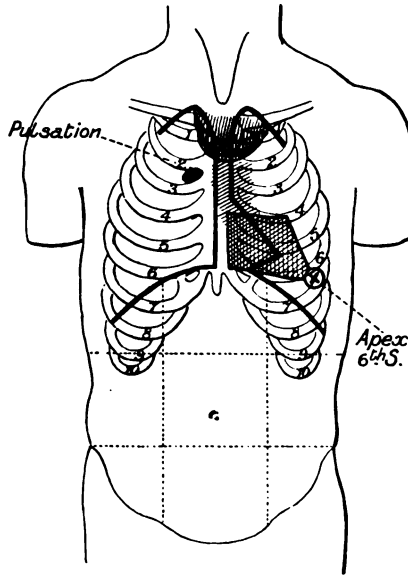
May, 1902. Patient has been lost sight of for the present.

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CASE 2.—F. M., æt. 33, male, admitted July 6th, 1901, under the care of Dr. Perry, for cough and dyspnoea. *Personal history*.—Patient was in the Service seven years, and had ague and fever in India, bronchitis for the last three years and a half. He has been in Charing Cross Hospital four times, with similar attacks to the present. These spasmodic attacks last

one hour, during which he is seized with a fit of coughing and difficulty of breathing, often brings up what he describes as "a lump of hard stuff," after which he feels better; the first attack began three and a half years ago, the present one eight weeks, during which he complains of having pain between the shoulders.

*Condition on admission.*—Temperature 98.2°, pulse 88, respiration 24. Patient is well nourished and inclined to be fat; the veins over the chest are very well marked; patient orthopneic. *Circulatory system.*—Pulses equal. *Heart.*—Impulse in sixth space, one and a quarter inches external to nipple



line and is diffuse, dulness begins at the fourth rib and left sternal margin, extending upwards towards the right third costal cartilage; some slight pulsation in the second right space; heart-sounds normal. *Respiratory system.*—Voice husky, has been so since first attack. *Larynx* slightly swollen and red, the right cord moves fairly well, the left not at all. *Chest* moderately distended, moves equally, a good resonant note obtained all over, respiration prolonged louder and harsher on the right side, râles and rhonchi heard all over chest. *Urine*, pale yellow; sp. gr. 1010; urea, 1 per cent., acid, no albumen. *Pupils* equally react to light and accommodation.

*Progress and treatment.*—July 5th. Amyl. Nitrite Capsules, 1 p.r.n. Tr. Lobelia Aetheris, m. 10. Liq. Trinitrini. m. 1. Tr. Belladonnæ, m. 6. Aq. Chloroformi, ad. 1 oz. every six hours.

July 8th. Patient had a very severe attack to-day lasting till the afternoon; relief was obtained by inhalations of chloroform.

July 14th. Another dyspneic attack, lasting two or three hours; complains of tenderness over upper part of chest.

July 15th. Another attack, lasting some hours, relieved by chloroform inhalations.

July 16th. Another attack, commencing at 5 a.m.; during this his lips became blue and face congested. Another attack at 7 p.m., lasting one hour, relieved by chloroform. Another attack at 2 a.m., after sleeping two hours.

July 17th, 11.30 a.m. *Injection of gelatin*, 240 c.c. (2 per cent.) into left side; at 6 p.m., temperature rose to 100.4°, patient not feeling so distressed, urine showed presence of precipitate with picric acid.

July 18th. Patient feeling better, cough not so troublesome.

July 19th, 6 p.m. *Injection of gelatin*, 250 c.c. (2 per cent.) followed by no rise in temperature; urine showed presence of precipitate with picric acid.

July 22nd. Patient feeling remarkably well this morning, cough not so troublesome; he has not required chloroform for the last three nights.

July 25th, 12 noon. *Injection of gelatin*, 250 c.c. (2 per cent.) followed by no rise of temperature.

July 27th. Patient feeling very well, area of dulness much reduced, better air entry. 6.15 p.m. *Injection of gelatin*, 200 c.c. (4 per cent.) followed by no rise of temperature.

July 29th. No pulsation in the second right space.

July 30th, 6 p.m. *Injection of gelatin*, 200 c.c. (5 per cent.); temperature rose from 97° to 100.8° twelve hours after, area of dulness over sternum diminished.

August 2nd. *Injection of gelatin*, 200 c.c. (5 per cent.) temperature remains subnormal.

August 9th. Patient has been feeling exceedingly well the last few days. *Injection of gelatin*, 120 c.c. (5 per cent.) which caused great pain. I.M.H. administered. Temperature rose from 97° to 99.2°, cough typically brassy.

August 12th. Patient not so well, another attack and cough, for which three inhalations of chloroform were given; not able to get the hard pellets of mucus up.

August 15th. Another attack of coughing, during which he brought up a tough piece of mucus, much better since this. *Injection of gelatin*, 200 c.c. (2½ per cent.)

August 16th. Site of injection red, swollen, and painful to the touch. Temperature 100°.

August 17th. Temperature 100.4° still some pain and tenderness over site of injection.

August 20th. Patient not so well, œdema over site of injection. 3 p.m., complains of stiffness of the lower jaw, and has difficulty in swallowing. Patient got worse towards evening, the stiffness extending down the neck. 12 p.m., abscess opened, and a considerable amount of foul green pus evacuated.

August 21st. Tetanic convulsions came on at 1 a.m. Patient put fully under the influence of chloral, breathing worse and convulsions frequent, excited by the slightest stimuli, oxygen administered; patient got gradually worse and died at 11 a.m.

*Autopsy.*—Large aneurysmal sac, involving second part of arch of the aorta, five inches in length, pressing on trachea and right bronchus, but not eroding it, displacing œsophagus well to the left, so that it made a considerable bend round the sac. The innominate and left carotid came off



from the sac, so that bases of both arteries were involved. A great deal of matting of the tissues around the sac. Inside was a good deal of old fibrin formation, amounting in the posterior of sac to one inch in thickness; over the surface was some very recent fibrin formation amounting in some parts to about one-eighth to one-quarter of an inch in thickness.

*Report of bacteriological examination.*—Pus from abscess. Microscopical—crammed full of bacilli, mostly short, no streptococci.

*Broth.*—Two or three kinds of bacilli, also cocci, no streptococci.

*Agar.*—Ditto ditto.

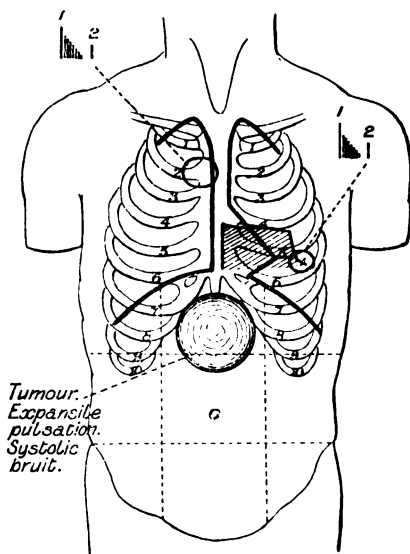
*G. F.*—No long bacilli, some like cocci.

*Plated.*—On anaërobic plate bacilli grew, which also grew well anaërobically no sporing bacilli survived 80° C. for half an hour.

The gelatin, incubated one week, showed sporing bacilli like tetanus, but after inoculation of 1 c.c. of gelatin into peritoneal cavity of a guinea-pig, gave no result.

CASE 3.—H. C., æt. 38, male, admitted July 19th, 1901, under the care of Dr. Perry, for pain in the loins. *Personal history.*—Patient is a married man, and has done a good deal of hard work, carting sacks of saccharine to brewery (three tons per diem), no syphilitic history. *Previous illnesses.*—Eighteen years ago had acute rheumatism. Two years ago he ruptured the muscles of his back, and was laid up for nine weeks. *History of present illness.*—Twelve months ago patient was seized with shooting pains in the back, he also noticed a throbbing in the abdomen; rest eases the pain.

*Condition on admission.*—Pulse 76. A strong healthy looking man, with a good deal of pulsation in the abdomen just below the epigastric angle, a soft pulsating ill-defined tumour can be made out, which extends from the ensiform cartilage to midway between it and the umbilicus, the outline is



more or less oval, dull to percussion, over it a soft blowing systolic bruit can be heard. *Circulatory system.* *Heart,* apex beat forcible, seen and felt in the fifth space half an inch external to the nipple, systolic bruit at apex, a faint but distinct one in the aortic area. Pulse irregular, rate 76. ? Water hammer in type. No tracheal tugging. Pupils equal and react. Urine, sp. gr. 1030, acid, albumen present in small amount.

*Progress and treatment.*—

July 19th. *Injection of gelatin*, 240 c.c. (2 per cent.) into left flank. Temperature rose to 99·6°.

July 22nd. Tenderness in region of injection.

July 25th. *Injection of gelatin*, 250 c.c. (2 per cent.) temperature rose to 100·6° at six p.m.

July 27th. *Injection of gelatin*, 200 c.c. (4 per cent.) temperature rose to 99° at 10 p.m.

July 30th. *Injection of gelatin*, 200 c.c. (5 per cent.) temperature rose to 100° next day; no albumen in urine now.

August 15th. *Injection of gelatin*, 200 c.c. 2½ per cent., injected into left flank. The size of tumour remains the same, throbbing less, and little pain now.

August 16th. Patient feels ill. Temperature 100·2°, severe pain in back, worse on movement.

August 18th. Patient despondent, pain in back worse, temperature 100·8°, site of injection red, swollen, and tender.

August 20th, 4 p.m. Patient complains of stiffness of the jaws, which has been getting worse for the last fifteen minutes. 5 p.m., tetanic spasms set in. 11 p.m., abscess at site of injection opened, foul green pus evacuated. Patient had to be kept under chloroform all night.

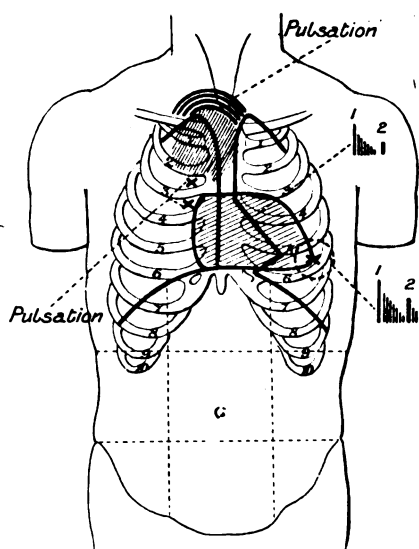
August 21st. Patient has a typical risus sardonicus, spine hyperextended, spasms are brought on by slightest touch, he sweats profusely and is able to swallow a little. Patient died at 2 p.m.

*Autopsy.*—Saccular aneurysm of the abdominal aorta, involving the cœliac axis, about two inches long by one and a half from before backward. The posterior wall is very thin, and closely adherent to vertebral column, so that it could only be separated from it with difficulty. The bodies of last thoracic and first lumbar vertebrae were considerably eroded, and formed the posterior wall of the sac. The interior surface of the sac was rough in parts, and showed patches of fibrin formation of a very flimsy and delicate nature, and no appreciable thickness.

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CASE 4.—A. D., æt. 45, female, admitted September 15th, 1901, under the care of Dr. Taylor, for pain in right arm and shoulder, and dyspnœa. *Personal history.*—Married, no children or miscarriages. *History of present illness.*—About a year ago patient noticed pain in the right shoulder for the first time; Christmas, 1900, it again appeared, and since then has never been long absent. Patient had bronchitis for six weeks at the beginning of 1901.

*Condition on admission.*—Temperature 97·4°, pulse 100, respiration 32. Patient somewhat orthopnœic, stout and well nourished. The face and neck are rather œdematous, especially on the left side; there is slight cyanosis and brassy cough. External jugular vein of right side felt engorged. *Circulatory system.*—There is a pulsating tumour in the supra-sternal notch, it measures



two inches from side to side, and about one inch vertically, there is also slight pulsation in the second and third right spaces, there is œdema of both breasts. *Heart*.—Apex felt in fifth space, one inch external to nipple, there is a short soft systolic at apex, accompanied by a diastolic, the former being heard all over the precordial area, the latter only at apex. Urine 1012, acid, no albumen; pupils equal and react; other systems normal. *Progress and treatment*.—Sept. 20th, Pot. Iod. 10 grains, t.d.s., pain confined to back of arm and hand, œdema unaltered, engorged veins seen in the lower intercostal spaces, *aneurysm with pressure on the superior vena cava diagnosed*.

September 27th. Œdema less, and aneurysmal swelling. Adde. Mist. Pot. Iod. 5 grains.

October 4th. Radial pulses unequal, right less than left.

October 7th. Swelling in episternal notch diminished.

October 7th. *Injection of gelatin* 250 c.c. (2 per cent.) into left groin. Temperature rose to 100° at 10 p.m.

October 11th. *Injection of gelatin* 250 c.c. (2 per cent.) into right flank. Temperature rose to 99° at 6 p.m. Headache.

October 14th. Pain in right shoulder and arm entirely gone.

October 15th. *Injection of gelatin* 250 c.c. (2 per cent.) Temperature rose to 99·4°.

October 16th. Soreness at site of injection with continuous headache.

October 18th. *Injection of gelatin* 250 c.c. (2 per cent.) into right thigh. No rise of temperature. Pot. Iod. 20 grains t.d.s.

October 19th. Dyspnoea more urgent and distressful.

October 20th. Patient in drowsy condition.

October 21st. Venesection 6 ozs. at 10 a.m., 11 a.m. patient comatose, oxygen administered; 5 p.m. patient improved, pulse at wrist stronger; brandy ordered.

October 24th. Patient continues to improve slightly.

October 25th. Œdema of hands, cyanosis of face, back and left side dry cupped.

October 31st. Upper extremities, neck and face and back of hands, very œdematous.

November 1st. Dulness over right chest, exploration, on aspiration three pints eighteen ozs. withdrawn, clear straw-coloured fluid.

November 2nd. *Injection of gelatin* 250 c.c. (2 per cent.), no reaction.

November 4th. Right eye closed, with œdema of lid.

November 6th. *Injection of gelatin* 250 c.c. (2 per cent.), no reaction.

November 8th. Air entering right chest better.

November 9th. *Injection of gelatin* 250 c.c. (3 per cent.), no reaction.

November 15th. Dulness of right chest increased, five pints six ounces of clear fluid aspirated.

November 23rd. Œdema of left hand more apparent.

November 24th. Patient got rapidly worse, more œdema, towards night became comatose.

November 25th. One a.m., venesection thirteen ounces.

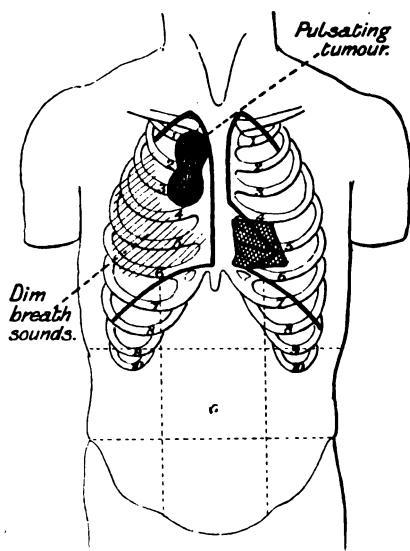
November 26th. Died 8 a.m.

*Autopsy.*—*Lungs.*—Right upper lobe compressed and airless, adherent to aneurysmal sac, left lower lobe compressed and airless; mediastinal glands enlarged. *Heart.*—Aortic valve thickened. *Aorta.*—Dilated and atheromatous. *Aneurysm.*—Eight by seven centimetres arises from the arch and reaches as high as the second tracheal ring, and is adherent to structures in front of trachea, opening into aorta about the size of a shilling, that into carotid, sixpence; about two-thirds of sac filled with laminated clot, fairly adherent to sac wall, the remainder was recent clot. *Kidneys.*—Tough and congested, small cyst on surface. *Spleen.*—Normal, two accessory spleens. *Liver and Intestines.*—Normal.

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CASE 5.—J. S., æt. 47, male, admitted October 23rd, 1901, under the care of Dr. Taylor, for pain and swelling on the right side of chest. *Previous illnesses.*—When twenty-five years old had rheumatism. *History of present condition.*—About fourteen months ago patient noticed that after exertion he had pain in the right side of chest. He was able to work up to Christmas, 1900, but since then unable to, through severe pain down the right arm and shoulder. During the last three weeks a swelling has appeared, and pain been more intense.

*Condition on admission.*—Temperature 96·2°, pulse 80. Patient orthopnœic, breathing quietly. There is a pulsating hour-glass-shaped tumour, extending above from the junction of the first costal cartilage with the sternum to the level of the third costal cartilage. It measures three and a half inches long, and about three inches across the upper end, two and a half inches round the constricted portion and two and three-quarter inches round the lower end. No bruit can be heard over it. *Circulatory system.*—Both radial pulses equal and regular. *Heart.*—Cardiac impulse can be felt in the fifth space in the nipple line, sounds normal. *Respiratory system.*—There is a deficient note over the right lung behind with diminished vesicular murmur, compensatory breathing over left lung. No tracheal tugging. Urine, sp. gr. 1030, acid; no albumen. Pupils equal and react.



*Progress and treatment.*—October 23rd, Pot. Iod. 15 grains t.d.s.

October 26th. Patient's cough of a brassy character, adde Mist. Pot. Iod. 5 grains.

October 28th. Complains of pain, cough troublesome, syr. codeinæ, syr. apomorphinæ, syr. pruni virginiani, a.s., one drachm every four hours.

November 2nd. *Injection of gelatin*, 250 c.c. (2 per cent.) into left side; temperature rose to 100° at 10 p.m. Patient skiagraphed and plaster cast taken.

November 6th. *Injection of gelatin*, 250 c.c. (3 per cent.) into right side, followed by no rise of temperature, tumour not changed in appearance.

November 9th. *Injection of gelatin*, 250 c.c. (3 per cent.) into left side; no reaction to it.

November 13th. *Injection of gelatin*, 250 c.c. (3 per cent.), no reaction.

November 19th. *Injection of gelatin*, 250 c.c. (3 per cent.), no reaction, no alteration in appearance of tumour.

November 25th. *Injection of gelatin*, 7.5 grams, slight reaction, patient is sure the tumour is smaller.

November 28th. *Injection of gelatin*, 7.5 grams, no reaction.

December 3rd. *Injection of gelatin*, 250 c.c. (4 per cent.)

December 9th. Physical signs unchanged, no alteration in tumour. Adde Mist. Pot. Iod. 5 grains.

January 27th. Thickness of aneurysm wall shows very little increase, pulsation about the same. Râles all over back and front of left lung; base of right lung dull from the eighth rib.

January 28th. Temperature 101.6°, pulse 120, respiration 40.

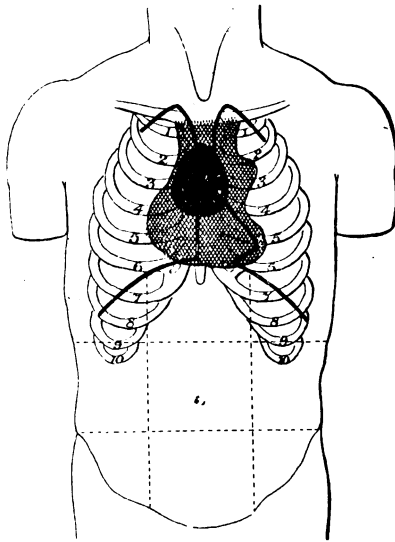
January 30th. Temperature 102.4°. Oxygen administered. Omit Pot. Iod.

January 31st. Temperature 98°8', respiration 44. Patient in low condition with laboured breathing. Base of right lung dull with deficient T.V.F. Râles over right and left lungs, oxygen every two hours.

February 1st. Dyspnoea increasing.

February 3rd. Patient died at 4.30 a.m. of gradual respiratory failure and heart's action.

*Autopsy.*—The aneurysm lay beneath the first, second and third intercostal spaces, and projected well over the middle line, bone eroded. *Heart.*—Weight 360 grams, pericardium normal, with no excess of fluid, some increase of sub-pericardial fat. Left ventricle hypertrophied, but muscle healthy, valves healthy. First inch of the ascending aorta showed signs of patchy atheroma, above this it is dilated into the sac of the aneurysm which extends up as far as the commencement of the innominate artery, and spreads towards the

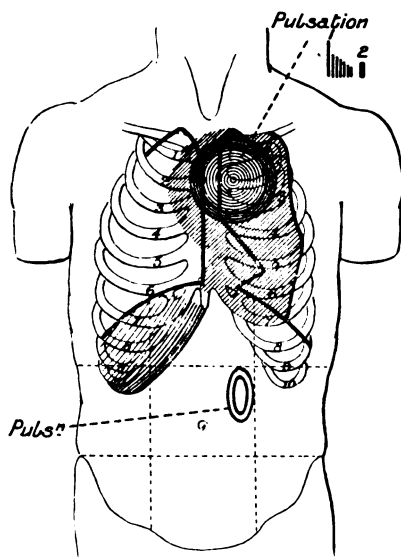


Skiagram November 2nd.

right. Anterior wall sac is filled with laminated clot, a half to three-quarters of an inch in thickness. *Lungs.*—A good deal of muco-pus in the trachea and bronchi, especially the right, which was compressed by the aneurysmal sac. Lobes of right lung adherent from old pleurisy, a good deal of pleurisy over the left lobe. Lungs œdematous with muco-pus in the tubes. Half a pint of fluid in the right pleural cavity. Condition of lungs one of septic broncho-pneumonia. *Kidneys* congested, capsule strips well leaving a somewhat granular surface. Cortex normal, in left kidney cortex more granular. *Liver*, weight 230 grams. Anterior border of right lobe adherent to diaphragm. *Intestines* normal. *Spleen*, weight 240 grams.

CASE 6.—R. C., æt. 45, male, admitted September 6th, 1901, under the care of Dr. Taylor, for dyspnoea and pain in left chest. *Personal history*.—Army schoolmaster, contracted primary syphilis at eighteen; had rheumatism. *History of present condition*.—Thirteen months ago felt pain of a dull aching character over heart, with numbness down the left arm.

July, 1900. Patient had a dyspnoic attack, pain ever since; dyspnoea has increased lately. *Circulatory system*.—Pulse only just felt at left wrist, stronger on right. *Heart*.—Apex felt in fifth space half an inch external to nipple line, pulsation can be felt over an area corresponding to first, second, and third left spaces, a systolic bruit is heard all over upper part of chest.



*Respiratory system*.—Slight tracheal tugging present; dulness all over left chest from clavical to seventh rib, vesicular murmur equal on both sides. *Liver*.—Edge felt half an inch below the costal margin, some abdominal pulsation. *Urine*.—Sp. gr. 1030, amber, acid, no albumen.

*Progress and treatment*.—

September 6th. Pot. Iod. 5 grains, t.d.s.

September 10th. Adde Mist. Pot. Iod. 5 grains.

September 21st. General condition better, very little pain. Left to come in again when gelatin was ready.

October 6th. Readmitted.

October 8th, 4 p.m. *Injection of gelatin*, 5 grams into abdominal wall. Temperature rose to 100° at 10 p.m.

October 9th. Temperature 100·2°, some tenderness of injection site.

October 11th, 6 p.m. *Injection of gelatin*, 5 grams. Temperature rose to 100° at 2 a.m. on the 12th.

October 15th, 7 p.m. *Injection of gelatin*, 5 grams. Pot. Iod. 20 grains, t.d.s.

October 18th, 6.30 p.m. *Injection of gelatin*, 5 grams, practically no reaction.

October 22nd, 6 p.m. *Injection of gelatin*, 5 grams. Temperature rose to 100.4° at 2 a.m. on the 23rd; no apparent improvement in aneurysm.

October 25th, 6 p.m. *Injection of gelatin*, 5 grams. Temperature rose to 99.2° next day.

October 30th. *Injection of gelatin*, 5 grams. Temperature rose to 99.2° at 6 p.m.

November 2nd. *Injection of gelatin*, 5 grams. Temperature rose to 100.8° at 10 p.m.

November 6th. *Injection of gelatin*, 7½ grams, no reaction.

November 9th. *Injection of gelatin*, 7½ grams, no reaction; ditto on 18th and 19th.

November 25th. *Injection of gelatin*, 7½ grams. Temperature rose to 99.4° at 10 p.m.

November 28th. *Injection of gelatin*, 10 grams, no reaction.

December 3rd. *Injection of gelatin*, 10 grams, no reaction.

December 7th. *Injection of gelatin*, 10 grams. Temperature 99° at 10 p.m.

December 9th. Condition unchanged; patient got up after tea. Systolic bruit heard behind on a level with spine of scapula.

December 12th. *Injection of gelatin*, 10 grams, no reaction.

December 15th. Patient discharged from hospital; condition practically unchanged.

February 14th, 1902. Patient caught cold and died of bronchitis at home. No autopsy.

CASE 7.—W. C. A., æt. 57, male, admitted Jan. 14th, 1901, under the care of Dr. Taylor, for cough and pain in chest. *Previous illnesses*.—Painter, thirteen years ago lead colic. *History of present illness*.—Four months ago cough, which has been worse for the last two, with pain in the chest. *Circulatory system*.—*Heart*.—Impulse not felt. *Pulses* equal, and not abnormal. There is some pulsation in the neck, heart-sounds normal. *Respiratory system*.—Respirations 35, some stridor, cough not clanging in character, brings up much mucus on coughing, and some hard masses, complains of pain in the left chest. There is paralysis of the left vocal cord, and well marked tracheal tugging. The inspiratory murmur is very harsh on the right side. *Pupils* equal and react. Other systems normal.

*Progress and treatment*.—Jan. 24th. Stridor not so marked. Pot. Iod. 10 grains, t.d.s.

February 1st. Patient vomited a dark coloured fluid.

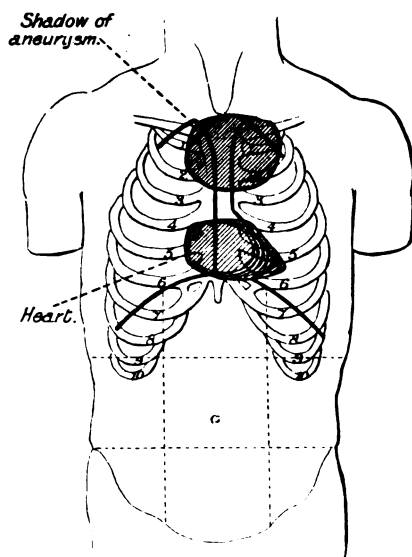
February 5th. Pain over fifth dorsal vertebra on pressure.

February 11th. Attacks of coughing frequent.

February 12th. Tenderness on pressure over second and third right costal cartilages, also fifth and sixth dorsal vertebræ.

February 19th. Patient a little better, coughing fits not so frequent, pain in chest fairly constant, and seems to be of a constricting character; at times he has very severe attacks of pain and becomes dyspnoic and cyanosed. Amyl. Nitrite capsules relieve this. For the last two or three days patient has had some hæmoptysis suggestive of a leaking somewhere. Pot. Iod., grains 15, t.d.s.





February 28th. *Injection of gelatin*, 3 ozs. (2 per cent.), into the abdominal wall. Temperature rose to 99°: some pain over site of injection. Patient skiagraphed. A dark shadow about the size of an orange was seen projecting to the left, and situated over the upper dorsal vertebræ.

March 2nd. Both vocal cords move to day. Patient refused further gelatin treatment.

March 8th. Patient was discharged at his own wish.

May, 1902. Patient has since been lost sight of.



# HEAT-STROKE.

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THE condition known as "heat-stroke" is well worthy of further study and investigation, not only on account of its practical importance, but also on account of the danger there is at the present time in the tendency to regard it as an infective disease, due to a hypothetical micro-organism. Its practical importance is contested by no one, for in many an army heat-stroke has been more fatal than the bullets of the enemy; year by year it takes toll, during the hot months, of civilians and soldiers, and is responsible for numerous cases of death or inefficiency among horses used for draught purposes or for cavalry. The view that "heat-stroke" is an infective disease is based upon insufficient grounds, and refuge should not be sought in a possible explanation by a hypothetical germ.

There is no intention to discuss here the contention of Dr. Sambon that "sunstroke is not heat fever, but an infective disease," and that "cases mentioned in England, France, Germany and Italy as "sunstroke" are always mistaken cases of syncope, delirium tremens, cerebral hæmorrhage, tuberculous meningitis, or cerebro-spinal fever." The question of diagnosis can be left to the physicians of those four countries. There are, however, in this condition of so-called "heat-stroke" certain physiological factors which are in danger of being overlooked. It is the object of this paper to consider these factors in relation to the causation and treatment of heat-stroke.

Heat-stroke has always been most prevalent among men exposed to a high temperature, and to the effects of muscular work. Since this is the case, the effects of the following factors

must be considered:—(i.) exposure to a high temperature; (ii.) muscular work, and (iii.) those conditions which render the effects of the previous factors more marked, namely, unsuitable clothes, heavy loads, and a moisture-laden atmosphere.

*The effects of exposure to a high temperature.*—Numerous observations show that healthy men exposed to high external temperature are able to so nicely regulate their temperature that it remains at the normal level. The experience of the inhabitants of tropical countries shows that it is possible to live, and even work in an atmosphere the temperature of which at times exceeds that of the body, and that the body is able by means of the cooling effect of the evaporation of sweat, to prevent its temperature rising a degree above the normal.

Much greater heat can be borne for short periods. Blagden and Fordyce observed their temperatures after remaining in heated rooms, and found that the effect varied according to the amount of moisture present in the air; thus, after they had remained fifteen minutes in a damp room heated to 129.9° F. (54.4° C.) the temperature of the mouth and urine was 100° (37.8°), but a similar exposure in a dry room, heated to 239.9°—260.0° F. (115.5°—126.7° C.), and in which beef steaks were being cooked by the heat of the air, did not raise the temperature of the body above the normal.

It is unnecessary to multiply the evidence upon this point, for it is generally admitted that a high external temperature alone will not raise the temperature of the body. The saturation of warm air with moisture will easily cause a rise in the internal temperature of man and animals by preventing the loss of heat, which in a dry atmosphere would be caused by the evaporation of water from the skin and respiratory tract. In any discussion of the prevalence of heat-stroke in different countries, the most important question is obviously not the temperature of the air, but the temperature of the air in relation to the amount of moisture and wind.

Exposure to a high temperature is not enough alone to explain "heat-stroke," and it is very doubtful if it alone ever caused "heat-stroke."

*The effects of muscular work.*—The exchange of material in the body is enormously increased by muscular work, and this involves far reaching effects on all the systems, for the body works as a whole. In order to supply the requisite energy for the muscular work, there is an exaggeration of the ordinary destructive side of the exchange of material, and this is shown chiefly by the great increase in the discharge of carbon dioxide and the absorption of oxygen. The oxidation is chiefly in the muscles, and the increased combustion is accompanied by an increase in the production of heat. In another paper evidence has been given that the internal temperature of healthy men may rise even as high as 101° F., owing to this increased combustion which is necessary for the performance of muscular work. There is no reason to look upon this moderate increase in heat as pathological or even wasteful, for those animals, namely, birds and mammals, which are characterised by an active life, have a high internal temperature. The chemical and physical changes involved in muscular contraction are facilitated, up to a certain point, by a high temperature. The normal temperature of birds is as high as 105·6° (40·9°), and even active insects, such as bees, are no exception to the general rule, for in their case the great production of heat during muscular work is masked by the great loss of heat from their small bodies which expose, relatively to their mass, an enormous cutaneous surface.

The increase in the discharge of carbon dioxide, and the absorption of oxygen necessitates greater activity of the lungs, and these organs in turn demand a more rapid supply of blood. For other reasons also the activity of the heart must be increased; the muscles require more blood, not only for the supply of material, but also for the removal of waste-products; the nervous centres demand more arterial blood and, if the muscular work be prolonged, the sweat-glands must be flushed with blood so that they may copiously secrete sweat, and thus assist in the discharge of any excess of heat. Increased activity of the heart is not the only requirement. An adjustment in the distribution of the blood must take place, and this demands the co-ordinated activity of the vaso-motor centres.

The inter-action between the heart and the lungs is very marked; increased respiratory movements assist the circulation of the blood by pumping blood from the venous system to the right auricle, and from the right to the left side of the heart. This function of the respiratory movements is often overlooked, but it is of great importance in connection with muscular work. Most athletes consider the dyspnœa observed during severe muscular work to be entirely a respiratory phenomenon; it is, they think, due to a want of oxygen and an accumulation of carbon dioxide. The relief which comes with the so-called "second wind" is on this theory the expression of the removal of these conditions. Experiments, however, upon the gases of the blood show no increase, but a decrease in the quantity of carbon dioxide, and no fall, but a rise in the quantity of oxygen during muscular work. The dyspnœa observed during exercise, especially in the case of untrained men, appears to be chiefly cardiac in origin. The muscles and the nervous system especially require during work an increased blood-supply; the heart, the blood-vessels, the lungs and the controlling nervous system respond to the demand, but at first fail to exactly adjust the balance. The right side of the heart is engorged, and until the lungs and the vaso-motor system adjust the supply of blood, dyspnœa and discomfort obtain. "Second wind" appears to be due to the adjustment of the vascular system. Increased frequency of respiration is also of value in increasing the loss of heat, for the expired air is saturated with moisture at the temperature of the body.

Stress is rightly laid upon this increased activity of the vascular system during muscular work, for in many cases of heat-stroke marked cardiac disturbance has been observed. Even apart from actual heat-stroke, it is probable that many of the cases of so-called "soldiers' heart" could be traced to the effects of muscular work under unsuitable conditions.

For severe muscular work it is not only requisite that the heart be healthy, but that the power of adjustment of the circulation by means of the vasomotor system and the pumping action of the lungs be efficient. The chief reason for the training

of men and horses for athletic contests is the need for the development and exercise of the power of regulation of the vascular system and of the temperature of the body. If all the systems of the body are to work to the best advantage, it is necessary that the temperature of the blood shall not exceed physiological limits.

*The effects of unsuitable clothes, heavy loads, and a moisture-laden atmosphere.*—It has been shown that economical muscular work demands the delicate adjustment of the chief systems of the body. It is to be expected, therefore, that even a healthy man may fail in this adjustment, if he be hampered by unfavourable external conditions. Unsuitable clothing is one of the most serious disadvantages with which the soldier has to contend.

Athletes are generally properly clothed during and after exercise. The soldier, left to the guidance of his instinct and common sense, clothes himself properly for the football or cricket-field; the garments allow freedom of movement and are scanty or loose enough to permit the ready discharge of the excess of heat produced during muscular work. The soldier on the march and under the orders and care of his officers is clothed in open defiance of common sense and physiological principles. His tunic generally fits as tightly as possible, is made of thick material and is fastened right up to the neck; his waist is hampered with a tight belt which interferes with abdominal breathing, and other straps supporting valise and haversack still further impede the movements of his limbs and body.

There is no valid excuse for the unsuitable clothes of soldiers. A gaudy, tight uniform is not necessary for the attraction of recruits or nursemaids; this is proved by the popularity of sailors and policemen with both classes.

*Heavy loads* make an extra demand upon the power of adjustment, even apart from the muscles directly involved. The weight in many cases disturbs the balance of the bilaterally symmetrical body, and to restore the equilibrium other muscles must be brought into play. Any weight, therefore, above that which is absolutely essential for the efficiency of the soldier as a fighting

man is uneconomical and must increase the number of men who "fall out" on a field-day.

Loads fixed by straps to the shoulders or waist must interfere with the respiratory movements and the proper ventilation of the clothes.

*A moisture-laden atmosphere* interferes with the regulation of the temperature of the body by diminishing the evaporation of sweat from the skin, and of moisture from the respiratory tract. In a consideration of the causation of "heat-stroke" this is as important a factor as the temperature of the air. Further, it is necessary to remember that soldiers marching in close order are in an atmosphere with a higher percentage of moisture, and often with a higher temperature, than that of the air outside their ranks. The soldier breathes air already warmed, fouled and saturated with moisture by the respiration and perspiration of his comrades. The condensation of the moisture forms a visible cloud on a cold day and can frequently be seen over a flock of sheep. Drovers know the danger of rapidly driving a flock of sheep in close order on a hot damp day; the cases of falling out and of death among sheep appear to be due to the same factors which operate in the case of man.

It is obvious that a stagnant atmosphere must render economical muscular work more difficult; a man has, other conditions being equal, greater difficulty in increasing his loss of heat. The effect of a wind in diminishing the oppressiveness of a stagnant atmosphere is too well known to need discussion.

These atmospheric conditions are important, for they help to explain the cases of heat-stroke in cities. Lofty houses arranged in streets render the air more stagnant, and by the absorption of heat and the radiation of one house against another make the air warmer, more stagnant and moisture-laden at night than the air of the open country around. These conditions must be considered together with the fact that normally the body shows a high temperature during the hours of activity and work, and a fall of temperature during the hours of sleep and rest. External conditions, therefore, which prevent the normal fall in the temperature as the hours of night draw on, not only cause discomfort



by preventing refreshing sleep, but place a strain upon the heat-regulating mechanism. These conditions will probably be found sufficient explanations for the fact that "heat-stroke" may occur at night.

The application of these principles may now be discussed. Muscular exercise increases considerably the internal temperature of men and horses and this occurs under physiological conditions. If the work be severe and be prolonged under unsuitable conditions the mechanism for the regulation of the temperature of the body will be taxed to prevent an immoderate rise. The subject may fail, owing to a hot moist atmosphere, to increase sufficiently the loss of heat by sweating, respiration, radiation and conduction. Sweating is a most effective method, provided that there is sufficient spare liquid in the body and the external conditions are favourable for the evaporation of the moisture. Farm-labourers work hard in the harvest-field when they are paid according to the work done; they are lightly clad, drink freely and sweat. Rarely are they overcome by the heat, although they are exposed for hours to the full glare of the sun.

Horses forced to do hard work in hot and moist atmospheres are very liable to "fall out" and often suffer from heat-stroke. This is the experience of the omnibus companies, even in a temperate climate such as that of England. The rectal temperature may be raised three or four degrees Fahrenheit by hard work (Hobday).

The horse regulates his temperature by sweating when he is hot, and this discharge of moisture necessitates the supply of water by the mouth. The efficiency of these horses is an important factor in the dividends of the companies. The horses are now watered, scraped and sponged, even when they are hot and sweating. The best proof of the value of such physiological treatment is that the companies find that it pays.

There is a widely spread belief that water is injurious to both horses and men when they are hot, and that even death has been caused by such a draught of cold water. These cases are not well authenticated; the subjects probably died because they were abnormally hot; not because they drank cold water. The

instinct and experience of animals and men lead them to drink when they are hot, and physiology shows that they are right.

Stress is rightly laid upon these cases of heat-stroke in horses, or this animal is often made, as is the soldier, to work hard under unfavourable conditions. The causes of inefficiency in the case of soldiers are constantly stated to be alcohol, nicotine and dissipation; none of these factors operate in the case of horses. The probability, therefore, of some healthy men failing under adverse conditions to prevent their temperature rising to a dangerous height must be granted. No doubt drunkenness and other vices would diminish the subjects' power of accommodation.

The question now arises whether the foregoing factors are sufficient to explain (i.) the origin and symptoms of heat-stroke; (ii.) the most effective treatment, and (iii.) the most efficient measures for the prevention of heat-stroke. The balance of evidence is in favour of an affirmative reply.

(i.) Origin and symptoms of heat-stroke. The prevalence of heat-stroke is always most marked when men and horses are obliged to work hard in a hot moist atmosphere. The muscular work necessarily increases the production of heat, and if the loss be not at the same time increased, the internal temperature must rise above the physiological limits. Any such abnormal rise damages the nervous system and the heart; the failure of proper nervous control over the mechanisms for the regulation of temperature will tend to cause a still further rise in temperature, for the warm-blooded animal now responds to changes of temperature in a manner similar to that shown by cold-blooded animals.

In some cases of heat-stroke the nervous symptoms are most marked, and in others the cardiac; the temperature is raised, and the skin is hot and dry. In so-called "heat-exhaustion" there may be a subnormal temperature, profuse sweating, marked cardiac disturbance and collapse.

These conditions can be explained without the aid of a hypothetical germ; the failure of the mechanism for the regulation of temperature may arise from lack of adjustment in the circulation, respiration, or excretory system, and will explain the ordinary

cases of heat-stroke with a high temperature and the cases of subnormal temperature in so called "heat-exhaustion."

(ii.) The most effective treatment of heat-stroke is one which deals with the case, not as an infective disease, but as a case of disordered or paralysed heat-regulating mechanism, in which the heart as a part of that mechanism may be involved. Cold baths will not lower, but raise the temperature of a healthy man, for his regulation is physiologically perfect. Cold water and free exposure do lower the temperature of patients suffering from heat-stroke, for their power of regulation is imperfect or partly paralysed. Under no other treatment do so many patients recover.

It is obvious that the disordered mechanism for the regulation of temperature may involve a subnormal temperature, as in cases of heat-exhaustion, and here the appropriate treatment is external warmth and stimulants.

(iii.) The most efficient measures for the prevention of heat-stroke are those which reduce the amount of work done and of heat produced by the body on a hot, damp day, or, this being impossible, increase the loss of heat. The War Office employs the first method by counter-ordering field-days when it is hot. This is efficient, but it is not training for war. The scientific enemy will compensate for the increased heat by increasing the capacity of its men to regulate their temperature. Light clothing, light loads, open order, a proper supply of water, and training on hot as well as on cold days will effect this.

The omnibus horses work hard even in hot weather; their efficiency is now increased by watering, scraping and sponging. The War Office trains its horses for war by stabling and feeding them in stalls, whereby their capacity for regulating their temperature is diminished. A scientific War Office, if such an institution be conceivable, would picket its horses, feed and exercise them under the conditions which obtain in war.



# NOTE ON THE EXCRETION OF UREA AND URIC ACID ON AN EXCESSIVE DIET.

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IN the course of a research on the metabolism of nitrogen and fat in forced feeding,<sup>1</sup> the opportunity was taken of determining in the urine the proportion of nitrogen which was excreted in the form of urea and of uric acid. It is desirable to put the figures thus obtained upon record, since so far as the writer has been able to ascertain, no account exists of the excretion of these bodies estimated by reliable methods for so long a period as that over which these observations extended, in a patient rapidly putting on weight and free from organic disease.

The publication and discussion of the results obtained has been somewhat delayed from various circumstances, not, however, without benefit, since our knowledge of the excretion of uric acid, and the relation which it bears to the other nitrogenous constituents of the urine, has been considerably added to during the past two years.

The patient was under the care of Dr. Hale White, to whom I am indebted for the opportunity of making this investigation, and for kindly obtaining information for me as to her subsequent progress. In a paper published in the *Journal of Physiology*<sup>1</sup> an account of the case will be found, with tables giving in detail the quantities and composition of the food taken and of the excreta.

<sup>1</sup> Hale White and Spriggs. *Journal of Physiology*, vol. 26, p. 151, 1901.

To avoid overburdening these pages only such of these particulars as are of importance for the present purpose are given here.

The patient was a woman of 38 years, whose weight under the stress of anxiety, sleeplessness and neglect of food had become reduced to 39·23 kilograms (six stone two pounds). In eight weeks, during seven of which she was kept in bed, she was fed up to a weight of 52·49 kilograms (eight stone four pounds). The diet was varied and, considering the patient's weight, enormous; the daily average of proteid taken was 243 grams, of fat 250 grams, in addition to abundant carbohydrates. The total energy of the food when expressed in units of heat was, on two average days, 5,300 and 5,600 large calories respectively. This gives 113 and 115 Calories for every kilogram of body weight. In a normal diet for a healthy man the proportion is about 43 Calories per kilogram, and under ordinary circumstances, any great increase of the quantity of food taken is resented by the digestive system. In a recent research on two normal men,<sup>1</sup> a diet containing 86 Calories per kilogram was reached in one individual. This could not, however, be tolerated for more than three days, owing to loss of appetite, feelings of distension, sleeplessness, dyspnoea, and finally diarrhoea and mucous colitis. Another man on a diet of 65 Calories per kilogram, suffered from similar symptoms after three days. The same workers, in their researches on metabolism in phthisis,<sup>2</sup> found that a diet containing more than 70 Calories per kilogram was attended with digestive disturbances in phthisical patients. Now, the patient under discussion absorbed the food given;<sup>3</sup> she suffered from no diarrhoea or disturbance of digestion, either during the period of observation or at the end of it; and she illustrates very well that people whose weight is below normal, and who are free from organic disease, can both take and make use of exceedingly large quantities of food. In this case, a diet nearly three times as plentiful as that of a normal individual was assimilated.

<sup>1</sup> Goodbody, Bardswell and Chapman. *Journ. of Physiol.*, vol. 28, p. 257, 1902.

<sup>2</sup> Goodbody, Bardswell and Chapman. *Medico-Chirurg. Trans.*, vol. 84, 1902

<sup>3</sup> Analyses of the *fæces* showed that over 96 per cent. of the nitrogen, and of the fat taken was absorbed.

## METHODS.

Since the hypobromite method of estimating urea gives a variable error, the Mörner-Sjöqvist method was used throughout; in this procedure hippuric acid is estimated together with the urea; but this takes little from the value of the results, as the proportion of hippuric acid excreted per diem in man is small compared with that of urea, being about 1 to 45 in a normal individual.<sup>1</sup>

The uric acid was determined by Hopkins' method.<sup>2</sup>

The figures obtained are given in the accompanying table.

Since the urine, preserved by chloroform, was analysed in two-day periods, the figures refer in each case to the excretion of 48 hours. It will be seen that on three days no analyses of urea are given.

The analyses of June 3rd are only those of the urine of 36 hours, and are not included in reckoning the averages.

## THE UREA.

The proportion of the total nitrogen in the urine which is excreted in the more highly oxidised form of urea is fairly constant in different individuals. Von Noorden gives from 82

<sup>1</sup> A modification of the method, with the object of avoiding the inclusion of hippuric acid, has recently been described. See Braunstein. *Zeitsch. f. Physiol. Chem.*, vol. 31, p. 381.

<sup>2</sup> Owing to the large quantities of urine passed by the patient the absolute amount of uric acid contained in 100 cubic centimetres (the quantity of urine which was taken for analysis) was sometimes very small. The writer has made some control estimations to find out how far this affects the accuracy of the figures obtained. From these it appears that when the absolute quantity of uric acid present in 100 c.c. is less than about .01 gram a result is arrived at which is too low. The liability to a relatively large error when working with too small quantities is of course common to all chemical procedures. Dr. Hopkins has shown that the initial precipitation of uric acid from urine by means of ammonium chloride is a complete one, and the shortage above mentioned is due to the subsequent manipulation of a precipitate containing less than ten milligrams of uric acid.

In cases of such great dilution, as has been pointed out to the writer by Dr. Hopkins, it is desirable to concentrate the urine on a water bath before making the estimation.

The percentage of uric acid found on June 17th, 19th, 21st, 23rd, and July 9th, was below .01, *e.g.*, on June 19th and 23rd, and on July 9th the percentages found were .006, .005 and .005 respectively. These dates are marked with an asterisk and are not included in calculating the average excretion of uric acid per diem.

TABLE I.

Date.	Total Nitrogen in Urine.	Urea.	Nitrogen as Urea.	Per cent. of total Nitrogen in Urine as Urea.	Uric Acid.	Nitrogen as Uric Acid.	Per cent. of total Nitrogen in Urine as Uric Acid.	Quantity of Urine in c.c.
June 3 (36 hrs. only)	—	28.22	13.17	—	.497	.166	—	2575
June 5	33.87	65.90	30.78	90.9	.657	.219	.647	5310
7	34.45	63.20	29.53	85.7	.595	.198	.575	4953
9	31.18	—	—	—	.764	.255	.818	5375
11	—	131.27	61.25	—	1.261	.420	—	9010
13	42.05	73.58	34.36	81.7	.468	.156	.371	5204
15	33.46	63.86	29.82	89.1	.753	.251	.750	5380
17	62.55	105.28	49.17	78.6	.544*	—	—	7770
19	37.07	65.22	30.46	82.2	.321*	—	—	5350
21	61.96	116.26	54.29	87.6	.546*	—	—	7803
23	43.47	81.57	38.09	87.6	.286*	—	—	5720
25	61.35	107.52	50.21	81.8	1.095	.365	.595	7820
27	48.44	89.35	41.73	86.1	.632	.211	.436	5746
29	64.08	—	—	—	1.036	.345	.538	7970

\* See Note 2, p. 273.



TABLE I.—continued.

Date.	Total Nitrogen in Urine.	Urea.	Nitrogen as Urea.	Per cent. of total Nitrogen in Urine as Urea.	Uric Acid.	Nitrogen as Uric Acid.	Per cent. of total Nitrogen in Urine as Uric Acid.	Quantity of Urine in c.c.
July 1	41.69	78.33	36.58	87.7	.745	.248	.595	5325
3	66.08	122.56	57.24	86.6	.902	.301	.455	8198
5	62.88	122.71	57.31	91.1	1.018	.339	.539	7831
7	68.67	136.05	63.54	92.5	1.128	.376	.548	8060
9	42.22	72.26	33.75	79.9	.287*	—	—	5887
11	68.31	132.20	61.74	90.4	1.164	.388	.568	7763
13	64.77	133.58	62.38	96.3	1.240	.413	.638	7748
15	—	127.16	59.38	—	1.085	.845	—	7897
17	41.41	76.04	35.51	85.7	.861	.287	.693	5743
19	43.49	89.75	39.11	89.9	.822	.274	.690	5136
21	48.99	98.29	43.57	88.9	.946	.315	.643	5256
23	51.96	81.33	37.98	73.1	1.011	.337	.649	5617
25	62.02	118.00	55.11	88.9	1.333	.444	.716	7841
27	22.85	38.07	15.44	67.6	.486	.145	.635	3117
Mean (2 days)	49.74	94.98	42.63	} 85.7 {	.904	.309	} .602 {	6928
" 1 day	24.87	47.46	21.31		.451	.154		3464

\* See Note 2, p. 273.

to 87 per cent.<sup>1</sup>; recent estimates by the Mörner-Sjöqvist method range from 82 to 96 per cent.<sup>2</sup>

The proportions in this case varied somewhat more widely about the same mean, forming from 96 to 73 per cent. of the total nitrogen. We may perhaps conclude that some instability of metabolism accompanied the phenomenal laying on of weight which was taking place, since the variations in the quantity of nitrogen which was being oxidised to urea showed a rather larger range than usual. In only three instances, however, is the figure under 80 per cent., with the exception of the last period when the food taken was being reduced.

Hopkins gives as the average proportion 86 per cent.<sup>3</sup>; in this case it was 85.7. Hence the patient on a greatly increased dietary, excreted on the whole the normal proportion of urea in relation to the total nitrogen of the urine.

#### THE URIC ACID.

On examining the figures of the uric acid analyses, the first point that suggests itself is that the excretion is irregular from day to day. In most instances this is due to differences in the quantity of urine collected in the two-day periods, owing to the urine not being passed at stated times.

It is also apparent that the excretion of uric acid was by no means great, the mean figure being .451 gram per day. A normal individual usually excretes a little over half a gram a day, but considerable variations in both directions, for instance from .2 to 1.4 grams,<sup>3</sup> occur under normal conditions. The output of nitrogen in this case was high, in accordance with the liberality of the diet, consequently the proportion of the total nitrogen which was excreted in the form of uric acid is made lower, being only .60 per cent. on the average, while the usual proportion varies from 1 to 3 per cent.

In order to understand why a patient having a large proportion of proteid in the food should excrete a small quantity of uric

<sup>1</sup> Lehrbuch, p. 63.

<sup>2</sup> Butler and French, these Reports, vol. lvi, p. 63.

Loewi. Arch. f. exp. Path. u. Pharm., vol. 41, p. 1.

<sup>3</sup> Schäfer's Text-book.

acid, we may look into the factors which are known at the present day to influence the excretion of this body.

Uric acid cannot be regarded alone since it forms but one of a group of chemically allied substances, variously known as the purin or alloxuric or xanthin bodies. At least ten of these have been found in the animal body. The more important besides uric acid are the four bases, xanthin, guanin, hypoxanthin and adenin.

When Kossel had shown that these bases could be derived in the laboratory from the nuclein contained in the nucleo-proteid of cells, and Horbaczewski had demonstrated the same for uric acid, feeding experiments were undertaken by various observers, which have established that the nucleo-proteids when taken into the body as food cause an increased excretion of uric acid.

The four bases above mentioned, besides others, are found in the tissues (and therefore exist in flesh foods) and in the urine; in the latter they are usually in smaller quantity than is uric acid, their proportion to that body varying from one-tenth to a quarter<sup>1</sup>; on a flesh diet they may equal the uric acid<sup>2</sup>. But reliable figures are scarce owing to the difficulty of accurately estimating them by hitherto available methods.

What is of most importance for the present purpose is to note that when given by the mouth, hypoxanthin,<sup>3</sup> xanthin, adenin, and probably guanin,<sup>4</sup> become oxidised and appear in the urine in the form of uric acid. But not entirely; Krüger and Schmidt recovered as uric acid 62 per cent. of the hypoxanthin given, 41 of the adenin and 10 of the xanthin. The last named, however, is not very soluble and was probably incompletely absorbed.

Now, since uric acid when given by the mouth appears in the urine as urea, some of the uric acid formed in the body by the oxidation of these bases will also form urea, and only what escapes this transformation can appear in the urine.

<sup>1</sup> Schreiber u. Waldvogel. *Arch. f. exp. Path. u. Pharm.*, vol. 42, p. 69, 1899.

See also Loewi. *loc. cit.* and Walker Hall. *Brit. Med. Journ.*, 1902, I., p. 1461.

<sup>2</sup> Loewi. *loc. cit.*

<sup>3</sup> Fischer. *Bericht. d. chem. Gesellsch.*, vol. 17, p. 329, and vol. 32, p. 434.

<sup>4</sup> Krüger. u. Schmid. *Zeitschr. f. physiol. Chem.*, vol. 34, p. 519, 1902.

Experimental evidence has been furnished<sup>1</sup> to show that it is the liver which oxidises uric acid (and purin bodies generally) to urea, and the proportion of these substances which is excreted in the urine is regarded as derived from that blood which circulates through the kidney before reaching the liver.

The results of feeding experiments therefore show that food gives rise to uric acid in the urine when it contains either purin bases, or nucleo-proteid, which is capable of giving rise to these bases.

But if food containing none of these substances is given, uric acid is still excreted and if no food at all is given there is a proportion of uric acid in the urine, the endogenous uric acid.

Little is known as to the origin of this part of the uric acid. It may be supposed that the body is in this case feeding upon its own flesh and excreting the uric acid, with a proportion of bases, derived from the breaking down of the nucleoproteid and the purin bases existing in the tissues, including the leucocytes. As to the amount produced in hunger, Succi, on the eighteenth day of his fast excreted .256 gram, and on the twentieth .244.<sup>2</sup> In two men, on the third day of hunger, the excretion has been found to be .197 and .205 gram.<sup>3</sup> Quantities as low have been observed in cases where, intentionally or not, the diet contained very little purin body producing substance. In Bonnani's case,<sup>4</sup> for example, .22 to .26 gram per day. The usual value for the whole endogenous purin bodies in the urine is, according to Burian and Schur,<sup>5</sup> .1 to .2 gram of nitrogen, that is, .22 to .45 gram of uric acid supposing three-quarters of the purin nitrogen is in this form. The value is said to be proportional to the body-weight.<sup>6</sup> Loewi points out that the total uric acid in the urine must not be regarded as the arithmetical sum of the endogenous uric acid, as ascertained during hunger, and that derived from the food, but as rather less than this sum,

<sup>1</sup> Burian u. Schur. *Arch. f. d. ges. Physiol.*, vol. 80, p. 335, 1900.

<sup>2</sup> Schreiber. *Ueber die Harnsäure*. Stuttgart. 1899.

<sup>3</sup> Schreiber u. Waldvogel. *loc. cit.*

<sup>4</sup> Bonnani. *Moleschott's unters. Naturl.*, vol. 17, p. 527.

<sup>5</sup> Burian u. Schur. *Arch. f. d. ges. Physiol.*, vol. 80, p. 335, 1900.

<sup>6</sup> Walker Hall *loc. cit.*

since the organism with a constant supply of nutriment would not use so much of its own material, and would produce therefore less endogenous uric acid.

It must also be mentioned that another source of uric acid production has been recently described,<sup>1</sup> namely, by synthesis from certain organic acids, *e.g.*, tartaric, lactic, and malonic, with urea.

The above considerations have furnished the explanation of the experiments of the last few years, which have abundantly shown that the uric acid excretion does not depend upon the amount of proteid in the food, except in so far as this proteid may contain bodies of the purin group, and have shown how valueless is the  $\frac{\text{uric acid}}{\text{urea}}$  ratio as a criterion of health. Sivén<sup>2</sup> excreted a constant amount of uric acid while varying his proteid ingestion from 18.5 to 145 grams in the day, of which 86 per cent. was animal proteid. What great variations of the  $\frac{\text{uric acid}}{\text{urea}}$  or the  $\frac{\text{uric acid nitrogen}}{\text{total nitrogen}}$  ratio must here have existed! He remained, however, in good health.

In the case of the patient with whom this note is concerned the food was analysed as regards the nitrogen and fat. Tables, giving the quantities of each foodstuff given daily, will be found in the paper cited.<sup>3</sup> No analyses were made of the purin contents of these foods, but with the help of the figures recently published by Walker Hall,<sup>4</sup> it is possible to form a rough estimate of the amount of these bodies which existed in the diet.

Only the daily average of the purin bodies which were given will be calculated in this way. It would be unjustifiable to apply such an inexact procedure with any particularity. It may be said, however, that the writer has constructed curves, showing the daily intake of the chief purin holding foods in the diet, such as meat and fish, and that they correspond roughly with the curve of uric acid excretion.

<sup>1</sup> Wiener. Hofmeister's Beiträge z. chem. Physiol. u. Pathol., vol. 2, p. 42.

<sup>2</sup> Sivén. Skand. Arch. f. Physiol., vol. 11, p. 123, 1900.

<sup>3</sup> Hale-White and Spriggs. Loc. cit.

<sup>4</sup> Walker Hall. Loc. cit.

The bulk of the proteid given was in the form of milk and eggs, which are free from true nucleoproteid or purin bodies,<sup>1</sup> supplemented by varied but not large additions of meat and fish.

In making the following calculation the main figures are taken from Walker Hall's analyses. Bacon is taken as having the same purin content as ham, lamb as mutton, and duck as chicken. Many kinds of fish were given. In those for which we possess estimates the purin bodies vary from .023 to .046 per cent. As salmon and halibut, which contain the higher proportion, were very little used, .03 is taken as an approximation or the present purpose. For French beans the analyses of haricot beans is used, which is certainly not understating the case. Peameal contains .016 purin nitrogen; green peas contain one-fifth of the proteid of dry meal,<sup>2</sup> and also lose extractives on boiling; hence the purin nitrogen in this food would not be more than .003 per cent.

The amounts of purin giving foods taken from June 3rd to July 28th, added together, were as follows:—

Food.	Total of grams taken.	Percentage of purin nitrogen in the food.	Remarks.	Total purin nitrogen in food.
Ham	261	.046	Walker Hall	.121
Bacon	416	"	do.	.191
Mutton	3020	.039	do.	1.178
Lamb	213	"	do.	.083
Chicken	1636	.052	do.	.851
Duck	100	"	do.	.052
Sweetbread	140	.402	do.	.563
Fish	5229	.03	do.	1.569
French beans	1101	.02	(Haricot)	.220
Peas	1399	.003	—	.042

The patient took also Benger's food, which no doubt contains some purin bodies, since its ferments are prepared from the pancreas. No allowance is made for this.

The sum of purin bodies arrived at is 4.870 grams, an average of .088 per day.

<sup>1</sup> Walker Hall found .0002 gram per cent. of purin nitrogen in milk.

<sup>2</sup> Hutchison. Food and Dietetics, p. 225.

But only a fraction of this can be expected to appear in the urine as uric acid.

Firstly, in cooking, either roasting or boiling, the purin bodies to a great extent escape. The loss is usually a half, and with boiling, may be two-thirds.<sup>1</sup> As regards the roasted food, the gravy was taken with the meat; with the boiled, however, and especially with the fish, there would be a smaller proportion of purin bodies than is given by the above figures. On the other hand, food loses in weight on both roasting and boiling, and since it was weighed cooked, the patient was receiving the equivalent of a rather larger proportion of uncooked food. But this would not be sufficient to counteract the loss of purin bodies in cooking.

According to Loewi,<sup>2</sup> the absorption of the nitrogenous part of the nucleoproteids is fairly complete. Probably, however, xanthin taken in the food is not completely absorbed,<sup>3</sup> and may constitute another source of loss of food purin.

Secondly, of the purin bodies which reach the blood, only a fraction (though a constant one)<sup>4</sup> is excreted as such in the urine; these substances, as above stated, are being constantly oxidised to urea, apparently in the liver, and only such portions as circulate through the kidney have an opportunity of being excreted without oxidation. Burian and Schur found that about half the purin bodies in meat appeared in the urine, and this is confirmed by Walker Hall, and the fraction found to be about the same for fish and fowl flesh.

Therefore, of the .088 gram of purin nitrogen in the food we cannot suppose that, after cooking and absorption, more than three-quarters, at the most, would reach the blood. Of this a half would be excreted as purin bodies in the urine, and of this half, a part would be turned out as bases, and not as uric acid.

About .03 to .04 gram is therefore an approximate figure for the daily amount of uric acid nitrogen in the urine to which the diet of this patient would give rise, or .09 to .12 gram. of uric

<sup>1</sup> Hutchison Food and Dietetics, p. 385.

<sup>2</sup> Loewi. Arch. f. exp. Path. u. Pharm., vol. 45, p. 147.

<sup>3</sup> *Vide Supra*, p. 277.

<sup>4</sup> Burian u. Schur. Arch. f. d. ges. Physiol., vol. 87, p. 239.

acid. The average daily excretion was .451 gram of this acid. This leaves for the endogenous uric acid about .33 gram per day, or allowing a margin for unreckoned purin bodies in the food, about .30 gram; this is a normal figure, especially when it is remembered that the patient's average weight was only 46 kilograms (seven stone three and a half pounds), and that she was resting, a condition in which the uric acid excretion has been found to be lower than in people who are up and about.<sup>1</sup>

An examination of the figures also indicates that this endogenous portion was rather larger during the last half of the period of observation, when the patient's weight was greater.

The writer would again emphasise what is indeed sufficiently obvious, that we are here dealing with an approximation, which will, however, illustrate the figures obtained<sup>1</sup> and indicate the way in which future more accurate work must be carried out.

It is evident from the above considerations that although the amount of food taken was very great, an examination of its constituents shows that the purin content was small, and hence explains the low uric acid excretion.

There is therefore no need to imagine that there was any retention of uric acid in this patient, neither did she suffer from the symptoms supposed to be associated with a diminished excretion of this substance.

The discussion of uric acid in relation to such symptoms can, however, be profitably deferred in view of the publication of further work which has been recently done in the laboratory.

<sup>1</sup> v. Siven loc. cit.



# THE EFFECT OF MUSCULAR WORK UPON THE TEMPERATURE OF MAN.

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1. Introduction.
2. Method of Observation.
3. Sources of error in determination of temperature in the mouth.
4. Temperature of the rectum, urine, and mouth, before and after various forms of muscular work.
5. Short account of previous work.
6. Summary and conclusions.
7. References.
8. Appendix.

1. *Introduction.*—The increased production of heat which accompanies muscular work is well known; a man walks more briskly if he feels cold, or, should he be obliged to remain in one place, “marks time” with his feet and vigorously throws his arms backwards and forwards across his chest. A horse willingly trots rapidly on a cold day. These voluntary means of increasing heat may be neglected or resisted, and then shivering, an involuntary muscular contraction, will occur. The sensation of warmth which accompanies the muscular work is not due only to the increased vascularity of the skin, acting upon the terminations

of the sensory nerves, but also to an actual rise in the temperature of the body. Shivering, which is accompanied by a contraction of the cutaneous blood-vessels, raises the temperature of the body, and, although at the time it is associated with a sensation of cold, is followed by relative warmth.

Although all physiologists recognise this increased production of heat during muscular work, some have yet maintained that the regulation of temperature is so complete that the internal heat of a healthy man rises but slightly or even falls during muscular work. Since this has been the teaching of many physiologists and physicians, the effects of muscular work in raising the temperature of the body has been to a great extent overlooked, or at least under-estimated. The cases of "falling out" and of "heat stroke" among soldiers, have been considered to be due to fatigue and a high external temperature acting upon a body debilitated by alcohol, dissipation, and nicotine. Recently a theory has been advanced by Sambon (1) that a hypothetical specific germ is the cause of "heat stroke." McCartie (2) and others (3) have rightly laid stress upon the importance of unsuitable clothes and heavy equipment. There is, as Riecke (4), Richet (5), and Ott (6) maintain, another factor, the rise of temperature, and it is a considerable rise, which is produced by muscular work.

The object of the investigation which forms the subject of this paper, was to determine the effect of muscular work upon healthy men, taking muscular exercise, but unhampered by unsuitable clothes or a heavy load. The effect of those two conditions will be considered in a subsequent paper.

2. *Method of observation.*—The deep temperature was measured by determinations of the temperature of the rectum or of the urine; observations were also made upon the temperature of the mouth, but for the reasons which are given later, we do not consider the results to be an exact measure of the internal heat of the body.

The clinical thermometers were verified at the Kew Observatory; the bulbs were 1.5 cm. long, and 2 mm. in diameter. During an observation the thermometer was retained in the rectum from two to four minutes, or the bulb was placed in the

stream of urine. The latter method<sup>1</sup> is rapid and convenient, and long series of experiments (7) have shown that, after the rectal temperature, the internal heat of the body is best represented by that of the urine.

For the rapid determination of the temperature of soldiers. Haldane and Pembrey have used the following simple apparatus. (Fig. 1). It consists of a zinc funnel 9 cm. long, 8 cm. wide at

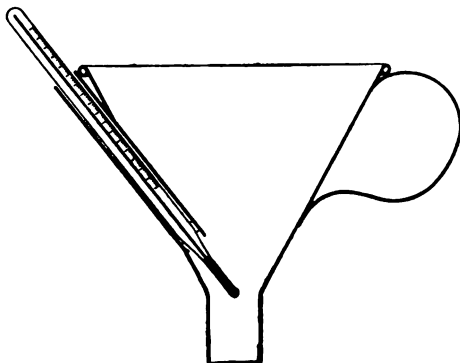


Fig. 1.—Vertical section of funnel with thermometer in position.

the base and 1.2 cm. wide at the apex; there is a side tube in which a clinical thermometer with a small bulb is placed so that the bulb projects into the apex of the funnel. The clinical thermometer (a so-called "half-minute thermometer") is fixed in position by two small pieces of rubber tubing, which at the same time prevent contact of the thermometer with the zinc. The stream of the urine is directed over the bulb of the thermometer and *not* down the sides of the funnel.

This method is the simplest and most exact method of rapidly taking the internal temperature of man. With two of these funnels Haldane and Pembrey were able to record the temperatures of eighty-three soldiers in one hour and fifteen minutes (page 304). The funnel is obviously not essential, but it enables the untrained man to direct the stream of urine more rapidly and surely over the small bulb of the registering thermometer.

<sup>1</sup> The temperature was taken in the urine by Stephen Hales, "Statistical Essays," London, 1731, second edition, vol. i., p. 59. Martine, "Essays, Medical and Philosophical," 1740, p. 335. Blagden, Phil. Trans., London, 1775, vol. lxx., pt. 1, p. 114. Davy, *Ibid.*, 1844, pt. 1, p. 63.

3. *Sources of error in determinations of temperature in the mouth.*—Observations upon the temperature of man are most conveniently taken in the mouth, but such determinations are not an exact measure of the internal heat of the body. A so-called “half-minute” clinical thermometer will not give true, or even comparable values for the temperature of the body under different conditions, even if it be retained for five or ten minutes under the tongue of a firmly closed mouth.

Since these statements are directly contrary to the opinion of some observers,<sup>2</sup> it is necessary to give the results of the experiments upon which they are based. Pembrey and Nicol (7) found as the result of sixty-three comparative determinations of the temperature of the rectum and mouth, an average excess of 0·65° C. (1·17° F.) in favour of the former, and a range from –0·33° to +2·56° C. (–0·59° to +4·59° F.); of these determinations only fourteen were made directly after muscular exercise, and the three or four cases, in which the temperature of the mouth was higher than that of the rectum, were those of observations made directly after a meal; the warm food and the increased vascularity and muscular activity of the mouth raised the temperature of that part. A comparison of twenty-two simultaneous observations of the temperature of the urine and mouth showed an average excess of 0·46° C. (0·83° F.) in favour of the former, and a range from –0·11° to +1·94° C. (–0·20° to +3·49° F.); only eight of these observations were made directly after exercise; in one observation taken directly after a meal, the temperature of the mouth was 0·11° C. (0·20° F.) above that of the urine. From these and other observations the conclusion was drawn that “the temperature of the mouth is not an exact measure of the deep temperature of the body; it is unreliable, especially after exercise or in cold weather, owing to the cooling of the mouth.”

Further comparative determinations of the temperature in the mouth, rectum, and urine, have been made, and the results are shown in the following table.

<sup>2</sup> Ringer and Stuart state that “due care being taken and sufficient time allowed, the temperature of the axilla is always identical with that of the mouth, and with that of the rectum four or six inches above its termination.” (Proc. Roy. Soc., London, xxvi., p. 186, 1877).

TABLE I.

	Excess of Rectal over Urinary Temperature.	Excess of Rectal over Buccal Temperature.	Excess of Urinary over Buccal Temperature.	Condition of Observer.
Average ...	of 14 = 0.32° C. (0.57° F.)	of 15 = 0.57° C. (1.03° F.)	of 11 = 0.80° C. (= 0.54° F.)	(P. R. Bolus). At rest indoors. At various times between 7.30 a.m. and 12.10 a.m.)
Maximum ...	" = 0.49° C. (0.89° F.)	" = 0.83° C. (1.50° F.)	" = 0.54° C. (= 0.97° F.)	
Minimum ...	" = 0.15° C. (0.27° F.)	" = 0.08° C. (0.14° F.)	" = 0.08° C. (= 0.14° F.)	
Average ...	of 20 = 0.38° C. (0.68° F.)	...	...	(P. R. Bolus). Taken at 8 a.m. after awakening.
Maximum ...	" = 0.51° C. (0.92° F.)	...	...	
Minimum ...	" = 0.29° C. (0.52° F.)	...	...	
Average ...	...	of 8 = 0.33° C. (= 0.59° F.)	...	(H. C. Lecky). Taken in bed at 7 a.m., November 2nd to 10th, 1899.
Maximum ...	...	" = 0.60° C. (= 1.08° F.)	...	
Minimum ...	...	" = 0.10° C. (= 0.18° F.)	...	
Average ...	...	of 6 = 0.18° C. (= 0.32° F.)	...	(H. C. Lecky). Taken in bed at 7 a.m., March, 1900.
Maximum ...	...	" = 0.31° C. (= 0.56° F.)	...	
Minimum ...	...	" = 0.05° C. (= 0.09° F.)	...	
Average ...	...	of 4 = 0.23° C. (= 0.41° F.)	...	(H. C. Lecky). Taken in bed at 7 a.m., October, 1900.
Maximum ...	...	" = 0.54° C. (= 0.97° F.)	...	
Minimum ...	...	" = 0.02° C. (= 0.04° F.)	...	
Average ...	...	of 5 = 0.51° C. (= 0.92° F.)	...	(H. C. Lecky). Taken at various times before exercise.
Maximum ...	...	" = 0.70° C. (= 1.26° F.)	...	
Minimum ...	...	" = 0.27° C. (= 0.49° F.)	...	

In these results there are included no observations made after exercise; the excess of the rectal temperature above the buccal temperature is, therefore, not so great as in the series by Pembrey and Nicol. The range is from  $0.83^{\circ}$  to  $0.02^{\circ}$  C. ( $1.50^{\circ}$  to  $0.04^{\circ}$  F.), and shows that true or comparable values for the temperature of the body cannot be obtained with a thermometer placed in the mouth.

The mouth is subject to considerable variations in temperature; the tissues forming its boundaries are relatively thin; evaporation of sweat from the skin, radiation from the vascular skin and cooling of the nasal and pharyngeal passages by respiration are factors which vary from time to time according to the temperature, moisture, and movement of the external air.<sup>8</sup>

When the subject is lying in bed the clothes around the neck maintain the temperature of the mouth at a value more comparable to that of the rectum. On the other hand, breathing through the mouth is imperative and natural in healthy subjects performing severe muscular work; thus the mouth is cooled by the inspired air and the evaporation of moisture and this cooling is not compensated by the warm expired air. Buccal respiration appears to play a part in the regulation of temperature during exercise; it increases the loss of heat. The temperature observed in the mouth in such cases is not a measure of the internal heat of the body.

4. *Temperature of the rectum, urine, and mouth, before and after various forms of muscular work.*—The observations were made upon ourselves; in some instances the muscular work was light, in others hard; in duration it ranged from four minutes to twelve hours. The table of results will show that in each case muscular work produced a rise in the temperature of the rectum and urine; the mouth showed sometimes a fall, sometimes a slight rise.

<sup>8</sup> These sources of error would appear to explain the subnormal temperatures observed in the mouth of healthy men living in the Alps. A series of observations by Buckmaster, Dent and Slater show that the buccal temperature is generally subnormal at high altitudes. These observations have not yet been published, but Dr. Buckmaster has kindly shown us the figures and allowed us to refer to them.

TABLE II.

Site of Observation.	Temperature before exercise. C.	Temperature after exercise. C.	Rise or fall in temperature. C.	Observer, nature of exercise, etc.
Mouth Urine Rectum	36.00° } 11.30 to 37.22°* } 11.41 37.67° } a.m.	35.45° } 1.2 to 37.94° } 1.12 38.00° } p.m.	-0.55° +0.72° +0.33°	M.S.P. A walk of about 4 miles on the Sussex Downs in a cold wind. No overcoat was worn. 26th December, 1897.
Mouth Urine Rectum	36.80° } 11.45 to 37.22°* } 11.57 37.55° } a.m.	36.32° } 12.52 to 37.83° } 1.8 37.94° } p.m.	-0.48° +0.61° +0.39°	M.S.P. Digging in the garden for 45 minutes. 27th December, 1897.
Mouth Rectum	36.44° } 5 p.m. 36.77° }	36.94° } 6 p.m. 38.27° }	+0.50° +1.50°	M.S.P. Walk for one hour. 4th September, 1897.
Mouth Urine Rectum	37.6° } 10.15 37.2°* } a.m. 37.6° }	37.3° } 11.45 38.0° } a.m. 38.1° }	-0.30° +0.80° +0.50°	M.S.P. After an easy bicycle ride for three and a half miles. Sweating profusely. Temperature of air 33.6° in the shade. There was a good breeze. 25th July, 1900.
Mouth Urine Rectum	36.89° } 3 p.m. 37.17° } 37.44° }	37.06° } 4.45 38.06° } p.m. 38.50° }	+0.17° +0.89° +1.06°	M.S.P. Work in the garden; no coat. Sun was shining, but there was a good breeze. Temperature of air in the shade = 15°. 20th April, 1901.

\* Only a small quantity of urine; too small for a correct determination of temperature.

TABLE II.—continued.

Site of Observation.	Temperature before exercise. C.	Temperature after exercise. C.	Rise or fall in temperature. C.	Observer, nature of exercise, etc.
Mouth Urine Rectum	... ... ...	37·11° 37·56°* 38·30° } 4.30 p.m.	...	M.S.P. Bicycle ride for 8 miles in 40 minutes. Sun shining. Temperature of air in shade was 19°. Body sweating, but not profusely; mouth feels dry. Only a small quantity of urine was passed. The thermometer in the mouth indicated 37·11° after it had been retained for 12 minutes, and 37·22° after 20 minutes. 16th August, 1897.
Mouth Urine Rectum	... ... ...	36·78° 37·56° 37·67° } 7 p.m.	...	M.S.P. Bicycle ride for about 2 miles in 15 minutes against a strong cold wind. Temperature of air = 5°. The temperature of the mouth was 34° directly after exercise.
Mouth Urine	... ...	36·95° 37·72° } 6 p.m.	...	M.S.P. Bicycle ride for about 2 miles in 13 minutes; no wind. Temperature of air = - 1°.
Mouth Urine	... ...	37·22° 37·89° } 3 p.m.	...	M.S.P. Bicycle ride for about 2 miles in 11 minutes; no wind. Temperature of air = 5°.
Mouth Urine	... ...	38·0° 38·0° } 8 p.m.	...	M.S.P. Sweating slightly after a slow bicycle ride for two miles. Temperature of air in shade = 27·75°. 20th July, 1900.

\* Only a small quantity of urine; too small for a correct determination of temperature.



TABLE II.—continued.

Site of Observation.	Temperature before exercise. C.			Temperature after exercise. C.	Rise or fall in temperature. C.	Observer, nature of exercise, etc.
Mouth	...	...	...	37·3° } 37·7°* } noon 38·0° }	...	M.S.P. Digging in the garden. Sweating. No coat, waistcoat open, no hat. Temperature of air in shade = 24·0°. 21st July, 1900.
Urine	...	...	...			
Rectum	...	...	...			
Rectum	...	...	...	38·33° 12·30 p.m.	...	M.S.P. Bicycle ride for about 16 miles in about two hours. Very hot day. Temperature of air = 30°. Sweating profusely. 13th August, 1900.
Mouth	...	...	...	36·94° } 37·70°* } 1·30 38·00° } a.m.	...	M.S.P. Walk for about 2 miles in 25 minutes. Comfortably warm in overcoat. Cold air, but no frost. 21st November, 1900. The temperature in the mouth was taken last, and the thermometer was retained for 10 minutes.
Urine	...	...	...			
Rectum	...	...	...			
Mouth	...	...	...	37·40° } 38·44° } 2·30 p.m.	...	M.S.P. Digging for about 2 hours. Sweating, at work in shirt sleeves. Temperature of air = 10°; damp. 25th December, 1900.
Rectum	...	...	...			
Mouth	...	...	...	36·60° } 38·35° } 11·30 38·70° } a.m.	...	M.S.P. Work for about 2 hours in snow. Temperature of air = 1°. Coat buttoned up. 5th February, 1901.
Urine	...	...	...			
Rectum	...	...	...			

\* Only a small quantity of urine; too small for a correct determination of temperature.

TABLE III.

Site of observation.	Temperature before exercise. C.	Temperature after exercise. C.	Rise or fall in temperature. C.	Observer, Nature of exercise, etc.
Mouth	...	36.22°	...	C.J.A.* Ascent of Eggner goch and little Allalinhorn. 4th August, 1897.
Rectum	...	38°	...	
Rectum	35.8° at 5 a.m. ...	39.4° at 11 a.m. 38.4° at 4 p.m.	+2.60°	C.J.A. Ascent of Mittaghorn (3148 m.) and Egghornhorn (3977 m.). Rectal temperature = 35.8° at 7 p.m. and 35.8° at 11 p.m. 7th August, 1897.
Rectum	36.11° at 3 a.m.	38.4° at 5 a.m. 38.4° at 8 p.m.	+2.29°	C.J.A. Ascent of Sud-Lenzspitze (4338 m.). 11th August, 1897.
Rectum	36.22° at 4—5 a.m.	38.4° at noon. 38.4° at 7 p.m.	+2.18°	C.J.A. Ascent of Simelhorn (2752 m.). Ascent took about 7 hours. Fine day, fairly warm. First climb of the season; out of training. Out for 14 hours, very tired, carried a camera. Arrived at hotel at 7 p.m. Rectal temperature at midnight was 36.89°. 17th August, 1898.
Rectum	36.33° at 3 a.m.	38.45° at noon. 38.4° at 7 p.m.	+2.12°	C.J.A. Ascent of Almagellhorn. Reached summit at noon, 8 hours from start. Weather fine and warm. Stiff work; out for 14 hours, very little food. Arrived at hotel at 7 p.m.
Rectum	36.89° early in afternoon	38.6 about 6 p.m. 38.5° 38.4° at 3 p.m.	+1.71°	C.J.A. After 6 hours walk. 21st August, 1898. After 8 hours' hard and rather dangerous climbing to the summit of Taschhorn (4498 m.). Rectal temperature = 36.5°. After a rapid descent over snow to the Kien Glacier. Rectal temperature = 38.4°. After dinner at Randa the rectal temperature was 36.89°, 3.30 p.m.. A very trying and fatiguing day.

\* This series of observations was cut short by the lamented death of Dr. Arkle in 1899. He took a great interest in the physiology of muscular work and training, for he was a good "all round" athlete and a member of the Alpine Club. The above observations were preliminary to a series of experiments, ended, alas! by an attack of influenza and pneumonia.

TABLE IV.

Site of observation.	Temperature before exercise. C.	Temperature after exercise. C.	Rise or fall in temperature. C.	Observer, Nature of exercise, etc.
Rectum ... Mouth ... Skin ... (abdomen)	37.27° } 36.44° } 36.30° } 11.35 p.m.	37.66° } 36.76° } 36.51° } 11.39 p.m.	+0.39° +0.32° +0.21°	P.R.B. A jump of 44 cms. in height was repeated as quickly as possible for 4 minutes. There was considerable sweat and a little dyspnoea.
Rectum ... Urine ... Mouth ... Skin ... (abdomen)	37.16° } 36.80° } 36.67° } 36.18° } 10 p.m.	37.95° } 37.37° } 37.13° } 36.52° } ...	+0.79° +0.77° +0.36° +0.34°	P.R.B. A sharp walk was taken. There was no dyspnoea; the mouth was kept closed. Pulse was 88 throughout the experiment. Temperature of outside air was 17.3°. 15th November, 1900.
Rectum ... Urine ... Mouth ... Skin ... (abdomen)	37.70° } 37.21° } 36.88° } 36.70° } 8.35 p.m.	39.03° } 37.65° } 36.33° } 36.08° } 10.10 p.m.	+0.33° +0.44° -0.55° -0.62°	P.R.B. A sharp walk was taken for 1½ hours. Temperature of outside air was 10.2°. Pulse 88 before, 88 after the walk. Respiration 23 before, 30 after the walk. Temperature of air of room was 13.1°. 16th November, 1900.
Rectum ... Urine ... Mouth ... Skin ... (abdomen)	37.53° } 37.21° } 36.83° } 36.85° } 2 p.m.	38.13° } 37.72°* } 36.67° } 36.21° } 3.45 p.m.	+0.60° +0.51° -0.16° -0.64°	P.R.B. After cycling at a good pace for about twenty miles. Temperature of air was 11.5°

\* Only a small quantity of urine; too small for a correct determination of temperature.

TABLE V.

Site of Observation.	Temperature before Exercise. C.	Temperature after Exercise. C.	Rise or fall in temperature. C.	Observer, nature of exercise, etc.
Rectum	36.75°	37.65°	+0.90°	<p>H.C.L. The temperatures before exercise were taken in bed at 7 a.m. immediately on being called. Rowing clothes were put on (shorts, woollen scarf round neck, vest, sweater) and a sharp run and walk taken for a little over a mile. Duration, never more than fifteen minutes. The temperatures after exercise were taken immediately on return.</p> <p>Average rise in rectal temperature of 8 expts. = 0.85° C. (1.53° F.)  max. = 1.00° C. (1.80° F.)  min. = 0.72° C. (1.29° F.)</p> <p>These observations were made on November 2nd, 3rd, 4th, 6th, 7th, 8th, 9th, 10th, 1899.</p>
Mouth	36.5°	...	...	
Rectum	36.8°	37.7°	+0.90°	
Mouth	36.6°	...	...	
Rectum	36.8°	37.6°	+0.80°	
Mouth	36.3°	...	...	
Rectum	36.75°	37.68°	+0.93°	
Mouth	36.51°	...	...	
Rectum	36.8°	37.52°	+0.72°	
Mouth	36.5°	...	...	
Rectum	36.8°	37.6°	+0.80°	
Mouth	36.7°	...	...	
Rectum	36.8°	37.55°	+0.75°	
Mouth	36.3°	36.75°	+0.45°	
Rectum	36.9°	37.9°	+1.00°	
Mouth	36.3°	...	...	

TABLE V.—continued.

Site of Observation.	Temperature before Exercise. C.	Temperature after Exercise. C.	Rise or fall in Temperature. C.	Observer, Nature of exercise, etc.
Rectum	36.55°	37.34°	+0.79°	H.C.L. Similar set of experiments to last.  Average rise in rectal temperature of 7 expts. = 1.01° C. (1.82° F.) max. = 1.56° C. (2.79° F.) min. = 0.78° C. (1.40° F.)  Average rise in buccal temperature of 6 expts. = 0.28° C. (0.50° F.) max. = 0.5° C. (0.90° F.) min. = 0.1° C. (0.18° F.)  These observations were made in March, 1900.
Urine	36.3°	—	—	
Mouth	36.3°	36.79°	+0.49°	
Rectum	36.55°	37.44°	+0.89°	
Urine	36.4°	—	—	
Mouth	36.5°	36.5°	+0.00°	
Rectum	36.55°	37.5°	+0.95°	
Mouth	36.4°	36.9°	+0.50°	
Rectum	36.33°	37.89°	+1.56°	
Urine	36.2°	—	—	
Rectum	36.55°	37.77°	+1.22°	
Mouth	36.4°	36.9°	+0.5°	
Rectum	36.61°	37.39°	+0.78°	
Urine	36.3°	—	—	
Mouth	36.3°	36.4°	+0.1°	
Rectum	36.72°	37.61°	+0.89°	
Urine	36.52°	—	—	
Mouth	36.52°	36.62°	+0.10°	

TABLE V.—continued.

Site of Observation.	Temperature before exercise. C.	Temperature after exercise. C.	Rise or fall in temperature. C.	Observer, nature of exercise, etc.
Rectum ...	36.70°	37.4°	+0.70°	<p>H.C.L. This series of observations was made Oct., 1900, in exactly the same manner as the last, but the exercise lasted not less than twenty-five minutes.</p> <p>Average rise in rectal temperature of 4 expts. = 0.99° C. (1.78° F.)  max. = 1.22° C. (2.19° F.)  min. = 0.70° C. (1.26° F.)</p> <p>Average buccal rise of ... 4 expts. = 0.43° C. (0.77° F.)  max. = 0.75° C. (1.35° F.)  min. = 0.23° C. (0.41° F.)</p>
Mouth ...	36.16°	36.61°	+0.45°	
Rectum ...	36.5°	37.61°	+1.11°	
Mouth ...	36.3°	37.05°	+0.75°	
Rectum ...	36.4°	37.35°	+0.95°	
Urine ...	36.4°	36.61°	+0.23°	
Mouth ...	36.38°	36.7°	+0.30°	
Rectum ...	36.55°	37.77°	+1.22°	
Urine ...	36.4°	36.7°	+0.30°	
Mouth ..	36.4°	36.7°	+0.30°	
Rectum ...	37.43°	38.10°	+0.67°	
Mouth ...	37.16°	37.30°	+0.14°	H.C.L. 25th December, 1900, 6 p.m. Twenty minutes' run and walk. Damp, warm evening.
Rectum ...	37.56°	38.10°	+0.54°	H.C.L. 29th December, 1900, 7.30 p.m. Fifteen minutes' run and walk. Dry and cold evening.
Mouth ...	37.05°	37.10°	+0.05°	
Rectum ...	37.50°	37.90°	+0.40°	H.C.L. 4th January, 1901. Ten minutes' run. Cold evening.
Mouth ...	36.90°	36.90°	+0.00°	
Rectum ..	37.50°	38.05°	+0.55°	H.C.L. 17th January, 1901, 6.40 p.m. Ten minutes' run. Warm and wet evening.
Mouth ...	37.80°	37.20°	+0.40°	
Rectum ...	36.60°	38.00°	+1.40°	H.C.L. 23rd February, 1901, 6 a.m. Forty minutes' run and walk in thick clothes.
Mouth ...	36.10°	37.30°	+1.20°	

The following series were made when the observer (H.C.L.) was wearing thick clothes. The individual experiments were, made under exactly comparable conditions—

TABLE VI.

Nature of Exercise.	Before Exercise.			After Exercise.	Rise in Temperature.
5 min. run ...	Mouth	97·16°	...	98·15°	0·99°
	Rectum	97·58°	...	99·00°	1·42°
10 min. run ...	Mouth	97·34°	...	98·45°	1·11°
	Rectum	97·80°	..	99·75°	1·95°
15 min. run ...	Mouth	97·43°	...	98·58°	1·15°
	Rectum	97·90°	...	100·50°	2·60°
20 min. run ...	Mouth	97·34°	...	98·96°	1·62°
	Rectum	97·70°	...	100·90°	3·20°
25 min. run ...	Mouth	97·52°	...	98·87°	1·35°
	Rectum	97·85°	...	101·50°	3·65°

TABLE VI.—continued.

Increment of work, as represented by 5 min. run, by healthy individual.	Rise of Temperature for each increment of work.	
5 min. ... ..	Rectal 0·53° F.	Buccal 0·12° F.
5 min. ... ..	0·55°	0·04°
5 min. ... ..	0·40°	0·47°
5 min. ... ..	0·45°	–0·27°

These results show that in the case of each observer muscular exercise produced a rise in the internal temperature, as observed in the rectum or in the stream of urine; the temperature of the mouth showed sometimes a rise, sometimes a fall. The maximal rectal temperature in each observer was above 38° (100·4°), and this temperature was accompanied by no distress or pathological condition.

It is necessary to point out that the rise in temperature is in some cases made more pronounced by the fact that the determination before exercise was made early in the morning, the time of the ordinary fall in the daily variation of temperature, whereas the observation after mountain-climbing was made later in the day, towards the time of the rise in the daily variation. Notwithstanding this, the observations show that two hours' climbing, even before 6 a.m. may raise the rectal temperature to  $38.4^{\circ}$  ( $101.12^{\circ}$ ). On those days when no climbing was done the temperature was low, in fact, showed a reaction towards a sub-normal temperature.

In any criticism the question of the physique of the observers must always be considered. The observers were healthy men between twenty and thirty-six years of age, they were accustomed to athletic exercise, and their medical training guarded against the oversight of any abnormal condition.

The series of observations made by Lecky, during training at Oxford, are of special interest; the exercise was similar in nature and duration each morning, and was taken under comparable conditions as regards time, clothing, and diet; the cold weather and the light clothing account for the fact that the rise in temperature was not greater, and the woollen scarf round the neck for the rise in the temperature of the mouth.

Three of the observers had been volunteers and had been impressed by the fact that exercise in uniform, and with the load of equipment and rifle caused a disproportionate amount of fatigue and discomfort. To test, therefore, the effect of exercise under favourable conditions the above series of observations were made.

The rise in temperature produced by muscular work must be considered physiological, and, if soldiers are to be properly trained to endure prolonged marches during hot weather, this increase in bodily heat must be taken into account in the regulation of clothing, load, the supply of water, and the frequency of halts. The series of observations by Haldane and Pembrey upon a company of regular soldiers at Aldershot show that the internal temperature of healthy men is higher than the normal generally given  $36.89^{\circ}$  ( $98.4^{\circ}$ ). The average temperature observed in the

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urine of eighty-three soldiers was  $37.58^{\circ}$  ( $99.64^{\circ}$ ), the maximum  $38.2^{\circ}$  ( $100.8^{\circ}$ ), and the minimum  $36.78^{\circ}$  ( $98.2^{\circ}$ ). These determinations were made between 3 and 4.30 p.m., after the mid-day meal and *not* after exercise.

5. *Short account of previous work.*—It is difficult to compare accurately the results which have been obtained by previous observers, for the temperature has been taken in different parts of the body. Some observers, especially Marcet and Lortet, found that muscular exercise produced a fall in temperature, others, Vernet and Forel in particular, always obtained a rise. The explanation of this want of agreement is to be found in the method of observation; our results show clearly that the temperature of the mouth is subject to considerable variations, and is not a true measure of the internal heat of the body; sometimes it indicates a rise, sometimes a fall in temperature after muscular work.

Although there are many isolated observations<sup>4</sup> upon the effect of muscular work upon the temperature of man, there are only a few series of records.

Marcet (8) made a series of observations upon his own temperature during Alpine climbing; by means of a mirror he observed the indications of a thermometer placed in his mouth when he was making the ascent of some of the Mount Blanc chain of mountains. Altitude alone had little effect upon the temperature of the body, but during the ascent the thermometer in the mouth invariably showed a fall, which was followed by a rise when rest was taken or the ascent made slowly. The mean result was  $35.8^{\circ}$  during an ascent and  $36.6^{\circ}$  during rest.

During two ascents of Mount Blanc Lortet (9) observed the temperature of his mouth; he always noted a fall in temperature; the lowest of sixteen readings was  $31.8^{\circ}$ , the highest  $36.3^{\circ}$ . If during the ascent a short rest were taken, the temperature of the

<sup>4</sup> See article "Animal Heat" by Pembrey; "Text-book of Physiology" edit. by Schäfer, vol. i.

See article "Chaleur" by Richet, *Dictionnaire de Physiologie*, t. iii., p. 99, 1898.

mouth rose, the maximum of sixteen observations during rest being  $37.0^{\circ}$  at Chamonix. These results were explained by Lortet to be due to a want of sufficient energy in the body to produce at the same time the great amount of work performed during the climb, and the heat necessary to maintain the normal temperature of the body.

The need of further experiments was recognised by Clifford Allbutt (10). By a series of observations he showed that Alpine climbing, even on a cold day, raised the temperature of the mouth about half a degree, the highest record was  $37.44^{\circ}$ . He maintained that it was necessary in order to obtain correct results to retain the thermometer in the closed mouth from fifteen to twenty minutes.

Forel (11) also found a rise in the temperature of the mouth both during the ascent and descent of a mountain; the mean rise during the former was  $1.34^{\circ}$ , during the latter  $1.54^{\circ}$ . He pointed out that the method of measuring the temperature in the mouth did not give a real value for the heat of the body, and that the cold and dry air of the mountains favoured a rapid loss of heat from the skin of exposed parts of the body.

Liebermeister and Hoffmann (12) observed a rise of about one degree in the temperature of the axilla both during the ascent and descent of a mountain.

The most valuable series of experiments upon the temperature of the body during mountain-climbing are those of Vernet (13). He observed the temperature in the rectum and found an average rise of  $1.64^{\circ}$  during the ascent, and  $1.70^{\circ}$  during the descent; the maximum,  $38.96^{\circ}$ , occurred after the ascent of the Grands-Mulets, 3050 metres high, when the temperature of the air was  $9.7^{\circ}$ . The results obtained by Marcet and Lortet were, he maintained, due to the determination of the temperature in the mouth, and he showed that during a climb the temperature of the rectum was considerably above that of the mouth, but that a rest brought about a fall in the former and a rise in the latter. This series is so important and has been so overlooked and neglected that it is reproduced here.

ASCENT OF DOLE, 1680 METRES HIGH, BY VERNET.  
June 18th, 1885.

		External Air.	Rectum.	Fall in temperature.	Mouth.	Rise in temperature.
3.35 p.m.	Departure from Duillier.					
6.	Bauloz, 1,148 metres high, during ascent ...	10.5°	38.46°		36.70°	
6.7.	seven minutes after halt ...	...	38.45°	-0.01°	36.70°	
6.15.	fifteen minutes after halt ...	...	38.44°	-0.02°	37.20°	+0.50°
6.25.	twenty-five minutes after halt.	...	37.76°	-0.70°	37.17°	+0.47°
7.13.	Pâturage, 1,442 metres high, during ascent ...	9°	38.31°	...	36.80°	...
7.20.	seven minutes after halt ...	...	38.10°	-0.21°	37.31°	+0.51°
7.28.	fifteen minutes after halt ...	...	38.02°	-0.29°	37.30°	+0.50°
7.38.	twenty-five minutes after halt.	...	37.83°	-0.48°	37.30°	+0.50°
8.18.	Dole, 1,680 metres high, during ascent ...	13.5°	38.63°	...	37.21°	...
8.25.	seven minutes after halt ...	...	38.42°	-0.21°	37.69°	+0.48°
8.33.	fifteen minutes after halt ...	...	38.28°	-0.35°	37.71°	+0.50°
8.43.	twenty-five minutes after halt.	...	38.00°	-0.63°	37.52°	+0.31°

Ott (6) made a series of observations upon the rectal temperature of soldiers before and after drill; the exercise was generally light and in no case severe or prolonged. The men marched from the barracks generally at 6 a.m. and returned about 10 a.m. The temperature of the air was generally about  $12^{\circ}$  ( $53.6^{\circ}$  F.), and even at mid-day was on no occasion above  $20^{\circ}$  ( $68.0^{\circ}$  F.). A rise of temperature was observed after exercise in 125 cases, the mean being  $0.74^{\circ}$ , ( $1.33^{\circ}$  F.); in three cases there was a fall,  $0.45^{\circ}$ ,  $0.4^{\circ}$ , and  $0.55^{\circ}$ , and in one case no change in the temperature. The highest rise observed was  $1.8^{\circ}$  ( $3.24^{\circ}$ ), from  $36.4^{\circ}$  ( $97.52^{\circ}$ ) to  $38.2^{\circ}$  ( $100.76^{\circ}$ ); the highest temperature recorded after exercise was  $38.3^{\circ}$  ( $100.94^{\circ}$ ).

Mosso (14) found that muscular exercise caused a considerable augmentation of the internal temperature, but with training the increase was gradually reduced. Zuntz and Schumburg (15) observed a slight rise in the temperature of the urine after a march for nine to thirteen miles; a load caused the temperature to rise to  $38.0^{\circ}$  ( $100.4^{\circ}$ ), or even to  $38.9^{\circ}$  ( $102.02^{\circ}$ ).

6. *Summary and conclusions.*—The chief results and the conclusions drawn from the observations recorded in the body of this paper and in the appendix are as follows:—

1. The determination of the temperature of healthy men by means of a thermometer inserted under the tongue in the closed mouth does not give accurate or comparable values for the internal heat of the body, even if the thermometer be retained for five minutes. If the time of retention be increased, as some observers have suggested, to ten or twenty minutes, the result is still inaccurate, for, as Vernet has shown, the temperature may fall in the rectum and rise in the mouth during that period.

2. The rectal temperature is the only accurate measure of the internal heat.

3. The method used by Stephen Hales, namely, to observe the temperature in the stream of urine, gives results which in point of accuracy come next to rectal determinations.

4. Comparative determinations of the temperature of the rectum and mouth before exercise show that the former may be one or even two degrees Centigrade ( $1.8^{\circ}$  or  $3.6^{\circ}$  F.) above the latter;

the difference depends chiefly upon the cooling of the tissues surrounding the buccal cavity. This is naturally more marked in cold weather, but even in warm weather there is a loss of heat from the boundaries of the mouth by the evaporation of sweat externally, and of moisture in the respiratory tract.

5. Comparative determinations of the rectum and urine before exercise show that the former is always greater than the latter; the average excess in forty-seven comparative observations was  $0.34^{\circ}$  ( $0.61^{\circ}$ ). This difference is due in many cases to a small quantity of urine, insufficient to properly raise the temperature of the mercury in the bulb of the thermometer. It is obvious also that the urine begins to cool, chiefly by evaporation, directly it leaves the body.

6. Muscular exercise in the case of each observer caused a rise in the temperature of the rectum and urine; the temperature of the mouth showed sometimes a slight rise, sometimes a fall.

7. The rectal temperature of healthy men doing muscular work may rise to  $38.5^{\circ}$  ( $101.3^{\circ}$  F.) without causing distress or any pathological effects.

8. The average urinary temperature of eighty-three soldiers was  $37.58^{\circ}$  ( $99.65^{\circ}$ ); the maximum  $38.22^{\circ}$  ( $100.8^{\circ}$ ), and the minimum  $36.78^{\circ}$  ( $98.2^{\circ}$ ). The men were at rest and the time was between three and half-past four o'clock in the afternoon.

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## APPENDIX.

(A.) *The temperature of healthy soldiers. Observations by J. S. Haldane and M. S. Pembrey.*

The permission of the War Office was obtained for the determination of the temperature of a company of soldiers at Aldershot. The men were healthy seasoned regulars belonging to "E" Company of the 1st Battalion The King's Own Regiment. The observations were made between 3 and 4.30 p.m. on August 13th, 1900, about an hour after the men had finished their dinner. The temperature was determined in the urine by the method already described (p. 285). It was a hot day; the temperature of the air was 31.5° (88.70°). The men wore light khaki uniform of the pattern used in India; no work or drill had been performed since dinner. They entered into the spirit of the investigation with the utmost goodwill, and thus it was easy to determine the temperature of eighty-three men in about one hour and a quarter.

TABLE VII.

Urinary temperature of 45 soldiers taken by J. S. Haldane.				Urinary temperature of 38 soldiers taken by M. S. Pembrey.			
F.	F.	F.	F.	F.	F.	F.	F.
99.4°	99.9°	99.3°	98.4°	100.2°	99.5°		
99.7°	99.2°	99.6°	99.4°	100.2°	99.2°		
99.9°	99.6°	99.7°	99.2°	99.4°	100.0°		
99.7°	99.5°	99.1°	100.0°	100.2°	98.8°		
99.7°	100.4°	99.3°	99.5°	100.4°	99.7°		
100.2°	99.7°	100.0°	100.4°	99.1°	99.3°		
99.2°	99.3°	99.8°	100.1°	100.8°	100.4°		
99.8°	99.8°	99.8°	100.3°	99.9°	99.7°		
99.8°	99.4°	98.5°	100.4°	100.0°	99.1°		
99.4°	99.2°	99.8°	99.8°	99.1°	99.0°		
99.0°	99.5°	99.3°	99.9°	99.7°	99.3°		
100.7°	99.0°	100.1°	99.8°	99.3°	98.2°		
99.3°	99.6°	100.3°	99.7°	99.3°			
99.7°	100.8°	100.3°					
99.6°	99.1°	99.8°					

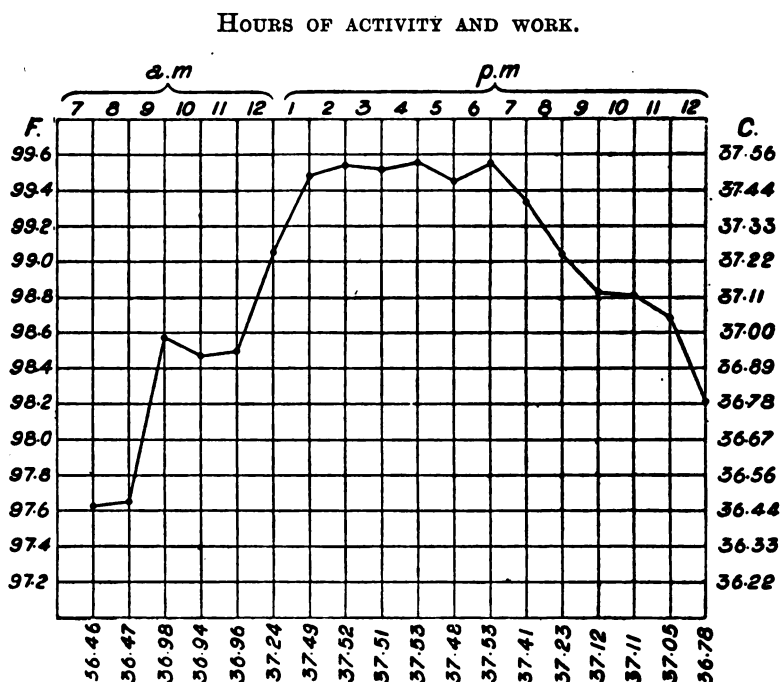
Average temperatures of 45 soldiers  
Maximum                   ...                   = 99.64°  
Minimum                   ...                   = 100.8°  
                                  ...                   = 99.0°

Average temperature of 38 soldiers  
Maximum                   ...                   = 99.65°  
Minimum                   ...                   = 100.8°  
                                  ...                   = 98.2°

The temperature of 21 men was as high as, or higher than 100°. In the case of the man who showed the minimal temperature, there was a good quantity of urine, more than enough for an accurate determination of the temperature; it was noted that this man was sweating profusely. The temperature of the air in the room was 89.7°.

(B.) *The daily variation in temperature. Observations by P. R. Bolus.*

The following is a chart showing the daily variation of Temperature of the Urine in the case of a medical student (P. R. B.), engaged in work at the Hospital and Medical School. The observations were made in October, November and December, 1900:—



Time of meals, 8 a.m., 1 p.m., 6 p.m., and variably 9:30 p.m.

Rest and sleep, 12 midnight to 7:30 a.m.

Weight of observer, 68.20 kilograms.



Time a.m.	7	8	9	10	11	12
No. of observations ...	21	20	10	12	9	13
Maximum ... ..	36·65	36·82	37·17	37·22	37·68	37·44
Minimum ... ..	36·28	36·36	36·31	36·81	36·89	36·77
Range ... ..	·37	·46	·86	·41	·79	·67

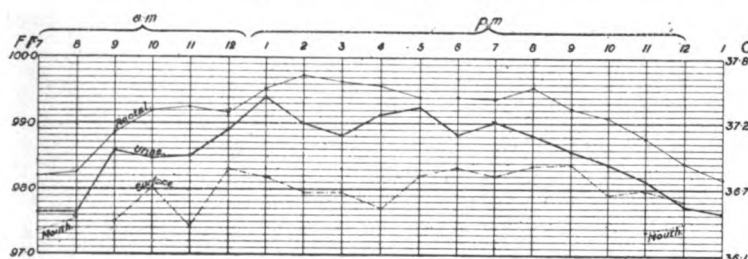
Time p.m.	1	2	3	4	5	6
No. of observations ...	16	19	22	17	25	12
Maximum ... ..	37·72	37·63	37·88	37·92	37·96	37·81
Minimum ... ..	36·95	37·21	37·40	37·19	36·88	37·24
Range ... ..	·77	·42	·48	·73	1·08	·57

Time p.m.	7	8	9	10	11	12
No. of observations ...	9	10	9	14	19	23
Maximum ... ..	37·93	37·50	37·32	37·64	37·48	37·30
Minimum ... ..	37·02	36·60	36·67	36·43	36·51	36·42
Range ... ..	·91	·90	·65	1·21	·97	·88

Maximum 37·96, 5.30 p.m. after 3 hours' walking. Minimum 36·28, 7.20 a.m. External temperature 12·1.

Temperature of the body at rest.—Observations by P. R. Bolus.



# URINE.

Time a.m.	7	8	9	10	11	12 noon.
Minimum ... ..	97·40	97·16	97·98	98·00	98·40	98·12
Maximum ... ..	97·95	97·97	98·80	99·03	98·90	99·51
Average ... ..	97·63	97·65	98·56	98·47	98·49	98·90

Time p.m.	1	2	3	4	5	6	7
Minimum ... ..	99·02	98·61	98·22	98·62	98·90	98·50	98·84
Maximum ... ..	99·70	99·75	99·75	99·70	99·81	99·21	99·62
Average ... ..	99·40	99·02	98·83	99·14	99·25	98·83	99·02

## URINE—continued.

Time p.m.	8	9	10	11	12 mid.	1 a.m.
Minimum ...	98·22	98·40	98·00	97·82	97·60	97·24
Maximum ...	99·73	79·52	99·02	99·00	98·36	98·02
Average ...	98·81	98·56	98·38	98·13	97·74	97·63

## RECTAL.

Time a.m.	7	8	9	10	11	12 noon.
Minimum ...	97·30	97·41	97·80	97·98	98·02	98·71
Maximum ...	98·58	99·00	99·98	99·54	99·60	99·52
Average ...	98·19	98·24	98·87	99·20	99·27	99·18

Time p.m.	1	2	3	4	5	6	7
Minimum ...	99·47	99·99	99·37	99·21	98·72	98·60	98·37
Maximum ...	99·59	99·04	99·80	99·75	99·74	99·73	99·80
Average ...	99·53	99·76	99·64	99·58	99·41	99·40	99·39

Time p.m.	8	9	10	11	12	1 a.m.
Minimum ...	98·98	98·40	98·42	98·31	97·62	97·31
Maximum ...	99·91	99·72	99·64	99·00	99·10	98·87
Average ...	99·57	99·22	99·07	98·78	98·40	98·17

## SURFACE.

Time a.m.	7	8	9	10	11	12 noon.
Average ...	35·32	35·59	36·40	36·69	36·36	36·84

Time p.m.	1	2	3	4	5	6
Average ..	36·77	36·63	36·64	36·50	36·79	36·85

Time p.m.	7	8	9	10	11	12 mid.
Average ...	36·78	36·86	36·88	36·62	36·65	36·56

*The variations are irregular, the range of variation being about 1° C. The temperatures (surface) for 7 and 8 a.m. are not printed in the chart.*

## MOUTH.

Time.	7 a.m.	8 a.m.	11 p.m.	12 midnight.
Minimum ...	97·19	97·22	97·16	97·00
Maximum ...	97·62	97·90	98·81	98·62
Average ...	97·37	97·56	97·34	97·48

The above records of temperature were taken between October 10th and December 30th, 1900.

The subject was at rest and had been so for not less than one hour before each observation.

The averages are struck from not less than eight readings, except in the case of surface temperatures taken between 8 a.m. and 1 p.m.

The time for rest and sleep was between 12 p.m. and 7.30 a.m.

The times for meals were 8 a.m., 1 p.m., 6 p.m., and variably 9.30 p.m.

The observations upon the temperature of the *urine* were made with a so-called "half-minute" thermometer, which was held in the stream very near the penis. In each recorded observation at least 100 c.c. of urine flowed over the bulb of the thermometer.

The *rectal* temperature was taken with a thermometer inserted 3 to 4 cms. into rectum and there retained for not less than three minutes.

The *surface* temperature was determined by means of a flat bulb thermometer placed on the skin of the abdomen at a point about one inch external to the umbilicus. The thermometer was held gently, and the reading was taken when the column of mercury was steady, usually after about two minutes.

Wool was worn next the skin.

The temperature of the *mouth* was taken when the bulb of the thermometer had been held under the tongue for not less than twelve minutes.

The number of observations in this series is as follows:—

Urine, 192.      Rectal, 170.      Surface, 115.      Mouth, 42.

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NOTE.—The special thermometers used in these experiments were supplied out of a grant from the Scientific Grants Committee of the British Medical Association.



**LIST**  
**OF**  
**GENTLEMEN EDUCATED AT GUY'S HOSPITAL**  
**WHO HAVE PASSED THE**  
**EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,**  
**&c., &c.,**  
**IN THE YEAR 1900.**

---

**University of Oxford.**

*Final Examination for the Degrees of Bachelor of Medicine and  
Surgery.*

H. S. French

|

R. C. Mullins.

*Intermediate Examination in Medicine.*

W. L. M. Day.

---

**University of Cambridge.**

*Degree of Doctor of Medicine.*

W. E. Alston.

|

E. A. Peters.

W. J. Harris.

|

A. E. Porter.

*Degree of Master of Arts (Honoris Causâ).*

F. G. Hopkins.

*Final Examination for the Medical and Surgical Degrees.*

**Part I.**

H. St. A. Alder.

| E. H. Kitchin.

| N. F. Ticehurst.

J. M. Brydone.

| J. S. Steele Perkins.

| E. G. Wales.

H. N. Clarke.

| E. W. S. Rowland.

**Part II.**

A. H. Davies.

| T. E. Holmes.

| J. G. Taylor.

G. S. Graham-Smith.

| F. J. Nicholls.

| L. Wilkin.

*Second Examination for the Medical and Surgical Degrees.*

PART I.

A. Morris.		B. H. Stewart.
------------	--	----------------

Part II.

S. W. Cole.		T. C. Lucas.
J. Goss.		C. M. Murray.

*Examination in Sanitary Science.*

Parts I. and II.

W. A. Densham.		J. V. R. Roberts.
----------------	--	-------------------

**University of London.**

*Examination for the Degree of Doctor of Medicine.*

E. Fisk.

*Obtained the number of marks qualifying for the Gold Medal.*

A. H. Carter.		H. E. Hewitt.
---------------	--	---------------

*Examination for the Degree of Bachelor of Surgery.*

Second Division.

J. A. Butler.		J. T. Dunston.		H. L. Eason.
---------------	--	----------------	--	--------------

*Examination for the Degree of Bachelor of Medicine.*

May.

First Division.

J. T. Dunston.

Second Division.

A. Fraser.		G. C. Owsley.
E. A. Miller.		F. D. Turner.

October.

Second Division.

J. A. Butler.		A. Densham.		P. T. Manson.
---------------	--	-------------	--	---------------

*Intermediate Examination in Medicine.*

January.

Entire Examination.

First Division.

K. Anderson.

Second Division.

H. Tipping.		W. E. J. Tuohy.
-------------	--	-----------------

*Excluding Physiology.*

Second Division.

A. W. Iredell.  
H. S. Jones.

G. H. H. Manfield.  
G. S. Robertson.

*Physiology only.*

First Division.

G. Evans.  
C. B. Penny.

E. F. Reeve.  
D. H. Trail.

F. C. Wetherell.

Second Division.

R. M. Barron.

A. W. Penrose.

July.

Honours Examination.

Robert P. Rowlands.

*Obtained the Gold Medal in Anatomy, and First Class Honours in Materia Medica and Pharmaceutical Chemistry.*

P. R. Bolus.

*Obtained the Gold Medal in Physiology and Histology, and Honours in Organic Chemistry.*

E. H. B. Milsom.

*Obtained First Class Honours in Physiology and Histology, and Honours in Organic Chemistry.*

E. G. Goldie.

N. Ivens Spriggs.

*Obtained Honours in Physiology and Histology.*

H. S. Brown.

*Obtained Honours in Materia Medica and Pharmaceutical Chemistry.*

Entire Examination.

First Division.

J. Braithwaite.

Second Division.

W. F. Box.

F. W. Fawcett.

L. S. H. Glanville.

P. W. Hamond.

R. Larkin.

L. H. Moiser.

F. C. Robinson.

B. H. Wedd.

*Excluding Physiology.*

First Division

C. D. Pye-Smith.

E. W. Strange.

Second Division.

G. B. F. Churchill.

H. M. Goldstein.

*Physiology only.*

Second Division.

F. A. Beattie.		J. Evans.		G. S. Robertson.
J. D. Bridger.		D. S. Graves.		

*Preliminary Scientific (M.B.) Examination.*

January.

*Biology.*

E. L. R. Norton.		W. P. Purdom.
A. B. O'Brien.		A. M. Roomes.

July.

Entire Examination.

Second Division.

E. H. Adams.		R. J. Bentley.		T. Turner.
G. N. Bartlett.		E. Bellingham-Smith.		H. A. Watney.

*Chemistry and Experimental Physics.*

I. R. Cook.		A. B. O'Brien.		J. T. Smalley.
H. S. Knight.		W. P. Purdom.		R. O. Williams.
E. F. Milton.		F. A. Sharpe.		

*Biology.*

T. H. Barton.		E. M. Harrison.		P. D. F. Magowan.
G. Hamilton.		E. C. Lowe.		

*Intermediate Examination in Science.*

Second Division.

A. G. Jones.

*Intermediate Examination in Science and Preliminary Scientific Examination conjointly.*

Examination for Honours.

R. W. Allen.

*Obtained Honours in Chemistry.*

F. M. Longson.

*Obtained Honours in Chemistry and Experimental Physics.*



**University of Durham.**

*Examination for the Degree of Doctor of Medicine.*

E. E. Frazer.

|

J. Harris.

*Examination for the Degree of Doctor of Medicine for Practitioners  
of Fifteen Years' Standing.*

W. H. Crosse.

|

R. A. Milligan.

*Examination for the Diploma in Public Health.*

A. J. Collis.

*Examinations for the Degrees of Bachelor of Medicine and  
Surgery.*

**Final Examination.**

S. C. Clapham.

|

H. C. Sturdy.

**Third Examination.**

G. W. Smith.

|

H. C. Sturdy.

*Obtained Honours.*

S. C. Clapham.

|

A. Reid.

**Second Examination.**

B. Glendining.

*Obtained Honours.*

E. G. Annis.

C. M. Anthony.

| J. W. Caton.

| A. Reid.

O. B. Travers.

| G. W. Smith.

| H. C. Sturdy.

**First Examination.**

B. W. Lacey.

|

A. V. Maybury.

*Elementary Anatomy, Chemistry, and Physics only.*

A. Reid.

**Royal College of Physicians of London.**

*Admitted to the Membership.*

N. D. Bardswell.

A. J. Cleveland.

|

T. D. Lister.

J. Lloyd Roberts.

*Examination for the Diploma in Public Health.*

J. Harris.

R. E. Williams.

*Final Examination for the License.*

January.

C. A. D. Bryan.	H. Durbridge.	O. Marriott.
H. N. Clarke.	R. Fell.	B. W. Moss.
A. H. Davies.	H. B. Foster.	W. R. E. Williams.

April.

J. B. C. Brockwell.	H. B. Dismorr.	L. C. Martin.
C. Edwards.	H. W. Fox.	E. A. Miller.
E. Cohen.	E. W. Goble.	R. C. Mullins.

July.

R. M. Barron.	R. W. B. Hall.	W. B. Secretan.
C. H. Brangwin.	E. F. Reeve.	T. P. Thomas.
J. M. Brydone.	A. Reid.	E. J. Tongue.

October.

H. Braund.	F. G. Gibson.	H. McD. Parrott.
J. A. Butler.	K. W. Goadby.	J. S. Steele Perkins.
P. W. L. Camps.	C. E. Hicks.	F. W. Sime.
E. H. Felton.	S. Hodgson.	L. E. Stamm.
D. Forsyth.	G. Lewin.	W. M. Thomas.
	T. J. Wright.	

**Royal College of Surgeons of England.**

*Final Examination for the Fellowship.*

L. H. McGavin.

*First Examination for the Fellowship.*

K. Anderson.	J. T. Hicks.	E. A. Peters.
A. R. Brailey.	A. R. McLachlan.	C. H. Robertson.
E. E. Henderson.	F. M. M. Ommanney.	Robert P. Rowlands.
	W. E. J. Tuohy.	

*Final Examination for the Membership.*

January.

C. A. D. Bryan.	H. Durbridge.	O. Marriott.
H. N. Clarke.	R. Fell.	B. M. Moss.
A. H. Davies.	H. B. Foster.	W. R. E. Williams.

**April.**

J. B. C. Brockwell.  
C. Edwards.  
E. Cohen.

H. B. Dismorr.  
H. W. Fox.  
E. W. Goble.

L. C. Martin.  
E. A. Miller.  
R. C. Mullins.

**July.**

R. M. Barron.  
C. H. Brangwin.  
J. M. Brydone.

R. W. B. Hall.  
E. F. Reeve.  
A. Reid.

W. B. Secretan.  
T. P. Thomas.  
E. J. Tongue.

**October.**

H. Braund.  
J. A. Butler.  
P. W. L. Camps.  
E. H. Felton.  
D. Forsyth.

F. G. Gibson.  
K. W. Goadby.  
C. E. Hicks.  
S. Hodgson.  
G. Lewin.  
T. J. Wright.

H. McD. Parrott.  
J. S. Steele Perkins.  
F. W. Sime.  
L. E. Stamm.  
W. M. Thomas.

*Final Examination for the License in Dental Surgery.*

**May.**

R. Westmore Allen.  
J. Bennett.  
E. Couchman.  
A. C. Edwards.  
P. H. Furnival.  
A. M. Gabriel.  
J. H. Greenwood.

W. Jarvis.  
L. C. A. Knight.  
A. L. Lambert.  
R. C. G. May.  
C. S. Morris.  
K. C. Ness.  
S. L. Prall.

R. B. Recordon.  
W. R. Searle.  
H. L. Shelton.  
A. M. A. Stevens.  
J. L. Wartski.

**November.**

A. W. Aldis.  
A. Archer.  
C. H. Aylen.  
K. Black.  
S. C. Bowle.  
F. W. Bromley.  
H. R. C. Butler.  
A. C. Carpenter.  
E. W. Corfe.  
A. R. Cummings.  
H. L. Dent.

A. Drewett.  
N. W. Green.  
P. Greenwood.  
C. Hickes.  
H. N. Hillier.  
J. J. Jiménez.  
G. F. Knowles.  
W. E. Meads.  
J. Milligan.  
F. Morris.  
J. W. Powell.

G. W. Ray.  
H. B. Ross.  
V. S. Sams.  
H. R. Shapland.  
C. R. Shattock.  
A. H. Smith.  
T. Walkington.  
D. H. Wallis.  
H. J. Webb.  
H. C. Winckworth.  
H. S. Wright.

**Society of Apothecaries of London.**

*Final Examination for the License.*

F. P. Bush.  
A. McC. Dallas.

F. R. Featherstone.  
T. E. Holman.

C. A. C. Salmon.

**MEDALLISTS AND PRIZEMEN.****JULY, 1901.***Open Scholarships in Arts.*

Herbert Orpe Brookhouse, Blackheath School, £100.

William Henry Dencer, Colfe Grammar School, £50.

George Cockcroft, Rossall School, Certificate.

Thomas Bramley Layton, Bradfield College, Berks, Certificate.

*Open Scholarships in Science.*

Richard William Allen, Guy's Hospital, £150.

Frederick Harold Lennox-Jones, Certificate.

*Scholarship for University Students.*

Arthur Robertson Brailey, Downing College, Cambridge. £25.

*Junior Proficiency Prizes.*

John Hunter Clatworthy, £20.

Henry Francis Bell Walker, £15.

Gerald Russell, £10.

Harry Hunter Carter, Certificate.

*The Michael Harris Prize for Anatomy.*

John Hunter Clatworthy, £10.

*Sands Cox Scholarship in Physiology.*

Henry Francis Bell Walker, £15.

John Hunter Clatworthy, Certificate.

Gerald Russell, Certificate.

*The Hilton Prize for Dissections (1900).*

Frederick Rogerson, £3.

Neville Ivens Spriggs, £2.

*The Arthur Durham Prizes for Dissection.**First Year's Students.*

Arthur Douglas Crofts, £5.

Arthur Boniface O'Brien, Certificate.

Frank Markland Longson, Certificate.

Howard Vincent Mitchell, Certificate.

*Senior Students.*

Edward Crosby Peers, £15.

John Edmund Spiller, Certificate.

John Alan Campbell Greene, Certificate.

*Dental Travelling Scholarship (1900).*

Frank James Pearce, £100.

*Dental Prizes.*

*First Year's Students.*

Herbert Theodore Binns, £10.

Wilfred Courtney Lyne, Certificate.

*Second Year's Students (1900).*

Ernest William Corfe, £15.

Harry Lambton Dent, Certificate.

William Edward Meads, Certificate.

*Second Year's Students (1901).*

Hubert Clarence Visick, £15.

James Bertrand Barron,	} equal Certificates.
Arthur Hastings Bell,	

*Practical Dentistry Prize.*

Herbert Theodore Binns	} equal £5 each.
Wilfred Ernest Griffin	

Edgar Everitt Lacey, Certificate.

*The Treasurer's Gold Medal for Clinical Surgery.*

Eccroyde Ihler Claxton.

*The Beaney Prize in Pathology (1900).*

James Alfred Butler, £34.

*The Golding-Bird Gold Medal and Scholarship in Sanitary  
Science.*

Sidney Thomas Reid, £20.

## THE PHYSICAL SOCIETY.

**Honorary President.**—SIR SAMUEL WILKS, Bart., M.D., LL.D., F.R.S.

**Secretaries.**—Mr. Bellingham Smith and Dr. Beddard.

### Presidents.

P. Turner, M.B., B.S., B.Sc., F. G. Gibson, M.A., F. G. Cross, F. O. Stöehr, B.A., M.B., B.Ch., H. S. French, M.B., B.Ch., F. Curtis, S. Hodgson, P. N. Blake-Ogders, B.A., D. G. Greenfield, M.B., C. H. Robertson, J. A. Butler, M.B., A. R. Thompson, P. R. Bolus, H. F. B. Walker.

### PRIZEMEN FOR THE SESSION 1900-1901.

Mr. F. Curtis was awarded the Society's First Prize of £10 for his "Notes from an Ornithological Cruise in the North Polar Sea," and the Second Prize of £5 was given to Mr. P. N. Blake-Ogders for his paper on "Jacksonian Epilepsy. Mr. H. S. French's paper on "The Treatment of Uræmia," gained the Treasurer's Prize of £5.

## CLINICAL APPOINTMENTS HELD IN THE YEAR 1900.

### HOUSE PHYSICIANS.

A. H. Carter. A. E. Clarke. J. F. Northcott.	H. L. Eason. J. G. Taylor. J. T. Dunston.	E. Fisk. W. G. Stewart. D. P. Watson.
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### HOUSE SURGEONS.

F. W. Brook. L. Wilkin. E. Cohen.	G. S. Simpson. A. G. Osborn. R. C. Mullins.	H. A. Gaitskell. C. T. Hilton.
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### ASSISTANT HOUSE SURGEONS.

G. M. Brown. E. E. Parrett. A. H. Davies. B. W. Moss. J. M. Bydone.	D. P. Watson. O. Marriott. E. W. Goble. E. F. Reeve.	C. B. Thomson. H. Durbridge. A. Fraser. T. E. Holmes.
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### RESIDENT OBSTETRIC ASSISTANTS.

F. W. S. Rowland. W. L. Baker. E. E. Parrett.	A. R. Thomas. A. R. McLachlan.	R. H. J. Swan. H. Durbridge.
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CLINICAL ASSISTANTS.

C. T. Hilton.	W. G. Stewart.	F. G. Cross.
A. G. Osborn.	A. H. Davies.	O. Marriott.
E. E. Parrett.	C. B. Thomson.	E. W. Goble.
B. W. Moss.	G. Lewin.	H. McD. Parrott.
T. J. Wright.	D. Forsyth.	T. P. Thomas.
T. E. Holmes.	J. S. S. Perkins.	J. A. B. Hammond.
P. W. L. Camps.	F. W. Sime.	M. D. Wood.
L. E. Stamm.	K. V. Trubshaw.	J. M. Brydone.

CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

E. A. Miller.	T. R. Beale Browne.	B. W. Moss.
J. C. J. DaSilva.	R. W. B. Hall.	G. Clarke.
C. H. Glenn.	F. W. Sime.	P. T. Manson.
A. Densham.	E. Shelton Jones.	S. Hodgson.
D. R. T. Griffiths.	R. S. Roper.	D. W. Smith.
R. Thompson.		

CLINICAL ASSISTANTS IN THE SURGICAL WARDS.

H. Bentley.	P. H. Ward.	H. B. Dismorr.
H. G. Rashleigh.	E. I. Claxton.	W. P. Ker.
L. Hirsch.	C. W. Hewlett.	T. B. Fawley.

SURGEONS' DRESSERS.

F. E. Welchman.	E. H. Kitchin.	F. D. Welch.
P. J. Nash.	H. A. Ehrlich.	D. R. T. Griffith.
F. Curtis.	E. I. Claxton.	G. G. Davidson.
L. E. Stamm.	E. C. Bevers.	D. W. Smith.
N. F. Ticehurst.	H. S. French.	H. K. Lacey.
R. P. Marshall.	F. W. Sime.	F. B. Manser.
P. T. Manson.	R. Thompson.	M. D. Wood.
R. Jiménez.	R. D. Smedley.	F. W. Smith.
T. H. F. Roberts.	J. F. Douse.	C. E. Gaitskell.
A. C. Nash.	E. Roberts.	G. S. C. Hayes.
W. M. Robson.	A. W. Gater.	A. W. Soper.
W. O. Roberts.	S. C. H. Bent.	L. Hirsch.
C. B. Penny.	E. F. G. Heap.	M. J. Rees.
J. E. Collins.	H. C. Keates.	E. Bigg.
F. D. S. Jackson.	F. A. Beattie.	G. H. Manfield.
M. A. Collins.	S. Child.	G. Evans.
R. C. Lawry.	J. A. Andrews.	F. C. Wetherell.
J. Evans.	H. P. Wiltshire.	E. G. Allport.
W. J. Davies.	H. W. Brown.	C. Tessier.
A. Wylie.	A. C. H. Gray.	V. M. Wallis.
G. T. Collins.	D. H. Trail.	C. H. Gask.
F. M. M. Ommanney.	A. D. E. Kennard.	H. Barber.
R. T. Collins.	H. A. Cutler.	A. H. E. Wall.
G. F. Humphreys.	W. G. Parker.	M. Coplans.
D. S. Graves.	B. Churchill.	W. H. Bowen.
F. Richmond.	J. W. Gromitt.	J. C. Bradbury.
C. R. Howard.	M. W. Cohen.	

## ASSISTANT SURGEONS' DRESSERS.

H. C. Keates.	J. F. Douse.	N. N. A. Houghton.
A. Wylie.	A. W. Soper.	W. M. Robson.
A. W. Gater.	G. S. C. Hayes.	S. C. H. Bent.
C. E. Gaitskell.	T. H. F. Roberts.	G. B. S. Soper.
F. Roberts.	A. Croneen.	A. C. Nash.
C. B. Penny.	M. J. Rees.	G. T. Collins.
F. C. Wetherell.	W. J. Davies.	J. Evans.
S. Child.	E. G. Allport.	J. A. Andrews.
E. Bigg.	H. J. Gater.	E. J. Crew.
G. Evans.	D. H. Trail.	H. M. Wiltshire.
A. C. H. Gray.	C. Tessier.	M. A. Collins.
H. W. Brown.	S. S. H. Shannon.	F. D. S. Jackson.
G. H. Manfield.	V. M. Wallis.	R. C. Lawry.
M. Coplans.	F. Richmond.	G. B. Churchill.
J. W. Gromitt.	J. C. T. Bradbury.	H. A. Cutler.
E. J. Crew.	J. C. Curtis.	A. D. E. Kennard.
R. T. Collins.	D. S. Graves.	A. H. Turner.
A. H. E. Wall.	M. W. Cohen.	W. H. Bowen.
H. Barber.	C. H. Gask.	W. G. Parker.
W. A. G. Stevens.	T. G. Miles.	G. Moir.
G. F. Humphreys.	W. W. C. Jones.	W. C. Lewis.
A. J. Urquhart.	C. E. Bartlett.	R. H. Terry.
F. M. V. Smith.	A. E. H. Pakes.	G. E. Malcolmson.
P. N. B. Odgers.	F. H. Parker.	F. L. Thomas.
A. J. Beadel.		

## DENTAL SURGEONS' DRESSERS.

E. Cohen.	E. A. Longhurst.	L. Hirsch.
A. C. Lewis.	R. C. Mullins.	L. Pern.
P. W. L. Camps.	E. G. Wales.	E. G. Andrews.
C. B. Thompson.	H. V. Bagshawe.	D. R. T. Griffiths.

## CLINICAL ASSISTANTS IN MEDICAL OUT-PATIENTS.

A. Fraser.	T. E. Holmes.	A. Densham.
T. E. Holman.	R. M. Barron.	W. M. Thomas.
P. S. Mandy.	J. S. S. Perkins.	L. E. Stamm.
R. P. Marshall.	J. A. Butler.	G. T. Willan.
R. D. Attwood.	L. Pern.	T. Morgan.

## DRESSERS IN THE EYE WARDS.

J. M. Brydone.	G. T. Willan.	F. G. Gibson.
E. F. Heap.	L. D. D. Cogan.	L. Pern.
R. S. Roper.	D. Forsyth.	P. W. L. Camps.
F. D. Welch.	F. G. Cross.	S. Ormond.
J. F. Robinson.	J. A. Butler.	G. B. Soper.
A. W. Soper.	A. H. E. Wall.	G. H. H. Manfield.
F. E. Welchman.	E. Stott.	
G. T. Collins.	J. D. Bridger.	

## DRESSERS IN THE THROAT DEPARTMENT.

R. M. Barron.	E. A. Miller.	H. Braund.
C. H. Glenn.	R. C. Mullins.	J. A. B. Brockwell.
W. B. Secretan.	F. D. Turner.	F. G. Cross.
K. V. Trubshaw.	F. G. Gibson.	T. P. Thomas.
C. B. Thomson.	G. Lewin.	F. W. Sime.
R. Tilbury.	G. Clarke.	T. H. B. Dobson.
J. A. Butler.	H. McD. Parrott.	J. F. Robinson.
J. S. S. Perkins.	H. Wachter.	F. J. Wright.



CLERKS IN THE THROAT DEPARTMENT.

A. Densham.	G. B. S. Soper.	C. H. Gask.
J. A. Andrews.	G. B. Churchill.	D. L. Morgan.
R. D. Attwood.	J. L. Whatley.	

MEDICAL WARD CLERKS.

A. C. H. Gray.	G. W. C. Hollist.	E. G. Allport.
H. P. Wiltshire.	J. A. Andrews.	G. B. Churchill.
D. S. Graves.	H. A. Cutler.	F. Richmond.
W. H. Bowen.	M. Coplans.	H. Barber.
M. W. Cohen.	A. H. E. Wall.	C. H. Gask.
R. T. Collins.	J. C. Curtis.	T. G. Miles.
J. W. Gromitt.	G. F. Humphreys.	W. A. G. Stevens.
G. Meir.	W. G. Parker.	J. C. O. Bradbury.
A. H. Turner.	R. Willan.	D. H. Trail.
A. D. E. Kennard.	G. Evans.	M. A. Collins.
F. C. Wetherell.	H. W. Brown.	E. Bigg.
W. J. Davies.	C. Tessier.	F. M. V. Smith.
C. E. Bartlett.	R. H. Terry.	A. J. Beadel.
G. B. S. Soper.	F. L. Thomas.	W. C. Lewis.
N. N. A. Houghton.	W. W. C. Jones.	P. N. B. Odgers.
A. E. H. Pakes.	G. E. Malcomson.	F. M. Smith.
F. H. Parker.	A. J. Urquhart.	F. J. Turner.
A. W. Iredell.	L. G. Nash.	H. T. Palmer.
W. H. Cole.	E. Faulks.	M. O. Wetherell.
J. D. Pearson.	T. M. Smith.	S. E. Prall.
C. J. Pinching.	O. B. Travers.	K. Anderson.
G. T. Wrench.	C. H. Robertson.	G. S. Robertson.
H. R. Grellet.	J. B. Copland.	C. M. L. Cowper.
W. E. J. Tuohy.	J. T. Hicks.	T. L. Pallant.
C. H. Denyer.	H. L. Shelton.	G. W. Smith.
A. P. Piggot.	W. L. M. Day.	R. Glendinning.
H. Tipping.	L. H. Moiser.	C. M. Murray.
E. H. Griffin.	R. C. Lawry.	

EXTERN OBSTETRIC ATTENDANTS.

H. S. French.	P. S. Mandy.	L. E. Stamm.
T. B. Fawley.	L. Hirsch.	D. W. Smith.
D. Forsyth.	J. F. Robinson.	P. W. L. Camps.
F. G. Gibson.	G. Graham Smith.	K. V. Trubshaw.
D. L. Morgan.	T. A. Matthews.	R. S. Roper.
E. G. Wales.	J. A. Butler.	B. Muir.
S. J. Ormond.	L. Pern.	T. H. B. Dobson.
G. Clarke.	F. W. Smith.	F. W. Sime.
F. B. Manser.	F. M. Ommanney.	R. Jimenez.
J. H. Frazer.	S. L. Prall.	F. Curtis.
E. C. Bevers.	J. A. B. Hammond.	S. Hodgson.
H. Wachter.	J. D. Bridger.	R. Thompson.
E. I. Claxton.	H. Davies-Colley.	N. F. Ticehurst.
E. H. Kitchin.	M. D. Wood.	H. K. Lacey.
H. Bentley.	G. G. Davidson.	P. D. Hunter.
E. J. F. Hardenberg.	S. C. H. Bent.	R. P. Marshall.
J. A. Wood.	W. J. Davies.	J. E. Collins.
J. Evans.	F. E. Welchman.	J. A. Andrews.
G. T. Collins.	M. J. Rees.	H. G. Rashleigh.
P. J. Nash.	A. W. Gater.	H. C. Keates.
A. C. Nash.		

## SURGICAL WARD CLERKS.

C. E. Bartlett.	F. L. Thomas.	N. N. A. Houghton.
W. W. C. Jones.	W. C. Lewis.	A. E. H. Pakes.
P. N. B. Odgers.	F. H. Parker.	A. J. Urquhart.
R. H. Terry.	A. J. Beadel.	F. M. V. Smith.
L. G. Nash.	F. J. Turner.	C. H. Bubb.
W. H. Cole.	A. W. Iredell.	J. D. Pearson.
M. C. Wetherell.	C. J. Pinching.	G. S. Robertson.
O. D. Travers.	H. R. Grellet.	C. H. Robertson.
W. E. J. Tuohy.	C. E. Adams.	K. Anderson.
J. E. Copland.	C. M. L. Cowper.	C. H. Denyer.
J. T. Hicks.	S. L. Pallant.	A. P. Piggot.
H. L. Shelton.	G. W. Smith.	H. Tipping.
W. L. M. Day.	B. Glendining.	L. H. Moiser.
C. M. Murray.	E. W. Strange.	C. H. Dawe.
J. Braithwaite.	F. W. Fawcett.	P. W. Hamond.
W. F. Box.	R. E. Brayne.	H. S. Brown.
W. H. Bush.	E. G. Goldie.	H. M. Goldstein.
H. S. Jones.	E. N. Jupp.	W. W. Read.
B. H. Wedd.	P. C. V. Bent.	G. L. Buckeridge.
C. F. Fraser.	E. J. Gaffney.	L. S. H. Glanville.
J. M. Bickerton.	J. H. Donnell.	R. Larkin.
B. B. Westlake.	C. M. Anthony.	R. G. Anderson.
N. I. Spriggs.		

## ASSISTANT SURGEONS' CLERKS.

K. Anderson.	H. Tipping.	H. S. Jones.
F. J. Turner.	G. S. Robertson.	W. H. Cole.
H. W. Brown.	G. W. Smith.	H. M. Goldstein.
T. G. Miles.	R. T. Collins.	G. B. Churchill.
A. H. E. Wall.	R. E. Brayne.	W. C. Lewis.
H. D. Smart.	E. H. B. Milsom.	A. M. Webber.
E. W. Strange.	J. S. Cooper.	C. D. Pye-Smith.
P. R. Rolus.	A. C. Osburn.	F. R. M. Knight.

## AURAL SURGEON'S DRESSERS.

R. C. Mullins.	T. E. Holmes.	E. A. Longhurst.
P. W. L. Camps.	H. Braund.	R. M. Barron.
T. J. Wright.	J. A. Butler.	E. G. Andrew.
D. L. Morgan.	R. P. Marshall.	E. Stott.
F. E. Welchman.	F. D. Welch.	R. S. Roper.
J. S. Perkins.	R. Thompson.	

## OBSTETRIC DRESSERS.

H. M. Reeve.	E. F. Reeve.	J. A. Wood.
E. Stott.	T. B. Fawley.	T. E. Holmes.
D. W. Smith.	D. L. Morgan.	H. McD. Parrott.
E. J. F. Hardenberg.	R. S. Roper.	F. W. Smith.
R. Jimenez.	J. D. Bridger.	E. I. Claxton.
T. R. Beale Browne.	N. F. Ticehurst.	J. L. Bates.
W. P. Ker.		

ASSISTANT PHYSICIANS' CLERKS.

C. Tessier.	S. Child.	E. J. Crew.
H. J. Gater.	L. J. Hughes.	G. H. H. Manfield.
W. J. Davies.	E. Bigg.	A. D. E. Kennard.
E. H. Felton.	J. N. Dyson.	W. A. G. Stevens.
T. G. Miles.	A. H. Turner.	G. Moir.
R. T. Collins.	C. E. Bartlett.	R. H. Terry.
P. N. Blake Odgers.	F. H. Parker.	W. H. Bowen.
S. L. Prall.	W. H. Cole.	H. T. Palmer.
C. J. Pinching.	L. G. Nash.	G. E. Malcolmson.
A. W. Iredell.	G. T. Wrench.	

CLERKS IN THE SKIN DEPARTMENT.

C. H. Glenn.	J. A. Andrews.	J. S. S. Perkins.
P. Mandy.	J. A. Butler.	F. G. Cross.
H. K. Lacey.	D. L. Morgan.	

POST-MORTEM CLERKS.

T. P. Thomas.	E. T. Jensen.	A. C. Lewis.
P. S. Mandy.	C. H. Glenn.	E. W. Goble.
W. O. Roberts.	E. F. Heap.	J. F. Robinson.
F. A. Beattie.	T. A. Matthews.	R. S. Roper.
J. M. Brydone.	P. W. L. Camps.	T. H. B. Dobson.
H. Davies-Colley.	N. F. Ticehurst.	H. Wachter.
R. D. Smedley.	G. Graham Smith.	F. G. Gibson.
C. D. Penny.	T. Morgan.	A. W. Gater.
E. J. F. Hardenberg.	R. Jimenez.	F. W. Smith.
W. A. G. Stevens.	T. G. Miles.	

CLERKS IN THE ELECTRICAL DEPARTMENT.

R. S. Roper.	D. Forsyth.	P. W. L. Camps.
P. S. Mandy.	D. W. Smith.	J. E. Collins.

CLERKS TO ANÆSTHETISTS.

J. A. Wood.	J. A. Butler.	G. T. Willan.
G. G. Davidson.	P. W. L. Camps.	T. P. Thomas.
F. W. Smith.	G. Clarke.	G. Graham Smith.
P. S. Mandy.	H. M. Reeve.	K. V. Trubshaw.
H. A. Higgins.	T. H. B. Dobson.	A. H. Davies.
H. McD. Parrott.	F. G. Gibson.	D. Forsyth.
J. F. Robinson.	R. S. Roper.	J. A. B. Hammond.
F. A. Beattie.	S. Hodgson.	N. F. Ticehurst.
E. Stott.	R. D. Attwood.	H. B. Carr.
E. G. Wales.	D. L. Morgan.	H. Wachter.
E. I. Davis.	J. D. Bridger.	E. C. Bevers.
L. E. Stamm.	E. G. Andrew.	F. E. Welchman.
P. J. Nash.	C. E. Gaitskell.	J. S. S. Perkins.
H. S. French.	F. D. Welch.	A. W. Soper.
A. H. E. Wall.	B. Churchill.	J. C. Curtis.
H. Bentley.	H. Davies Colley.	R. Thompson.
T. H. F. Roberts.	H. C. Keates.	D. R. T. Griffiths.
C. B. Penny.	S. C. H. Bent.	M. J. Rees.
H. W. Brown.	W. M. Robson.	G. S. C. Hayes.
G. T. Collins.	A. Densham.	J. Evans.
L. G. Nash.		

## DENTAL SCHOOL.

### APPOINTMENTS HELD IN THE YEAR 1900.

#### DENTAL HOUSE-SURGEONS.

J. Black.	H. T. Campkin.	F. J. Pearse.
R. B. Recordon.	J. Bennett.	C. S. Morris.

#### ASSISTANT DENTAL HOUSE-SURGEONS.

R. C. G. May.	S. L. Prall.	E. W. Corfe.
T. Walkington.	E. J. Gaffney.	J. J. Jimenez.
G. W. Badcock.	A. H. Clogg.	

#### DEMONSTRATORS IN THE CONSERVATION ROOM.

F. W. Bromley.	J. J. Jimenez.	H. J. Webb.
G. W. Badcock.	A. H. Clogg.	S. W. Iles.
S. C. Bowle.	E. L. Davis.	W. E. Griffin.
R. J. Green.	H. C. Visick.	J. W. Walton.

#### ASSISTANT DEMONSTRATOR OF DENTAL MICROSCOPY.

E. J. Gaffney.

#### DRESSERS IN THE EXTRACTION ROOM.

J. S. Francis.	J. Milligan.	G. W. Badcock.
E. L. Davis.	H. J. Corin.	A. H. Clogg.
W. E. Lowe.	S. R. Lidiard.	W. E. Meads.
G. F. Knowles.	D. H. Wallis.	O. Black.
C. A. Buckell.	G. F. Sargood.	S. J. Saunders.
W. K. Perry.	A. D. Knight.	W. E. Griffin.
S. Clifford.	A. H. Staple.	C. Mills.
G. H. Morris.	S. W. Iles.	F. G. Day.
W. Morgan.	C. H. Mason.	S. C. Bowle.
H. L. Whitlow.	T. C. Holford.	G. S. H. Barnett.
P. J. Reid.	H. C. Visick.	F. R. E. Palmer.
A. H. Bell.	L. Myer.	J. E. Spiller.
E. Phillips.	T. A. Chignell.	A. C. Stroud.
W. H. Peatfield	P. F. Minett.	A. E. Steele Perkins.
A. H. Forbes.	W. Giles.	H. D. Griffith.
C. D. Wood.	W. Henderson.	A. R. Beaumont.
J. B. Barron.		

#### DRESSERS IN THE GAS ROOM.

P. Greenwood.	J. E. Spiller.	A. C. Stroud.
J. H. Hinton.	E. Cock.	W. Henderson.
G. W. Ray.	H. L. Dent.	J. J. Jimenez.
V. E. Turner.	P. Greenwood.	F. W. Bromley.
A. R. Cummings.	H. Hatton.	C. B. Ross.
G. F. Knowles.	C. Hickes.	F. Morris.
R. S. Witcomb.	D. H. Wallis.	W. K. Perry.
A. B. W. Rust.	A. Drewitt.	V. S. Sams.
G. H. Morris.	A. H. Smith.	O. H. Dignum.
T. Robinson.	W. E. Meads.	C. S. Morris.
H. J. Webb.	K. Black.	H. C. Winckworth.
N. W. Green.	H. S. Wright.	E. J. Gaffney.
A. H. Clogg.	S. W. Iles.	J. W. Powell.
H. J. Corin.	W. E. Lowe.	G. W. Badcock.
R. S. Witcomb.	E. L. Davis.	J. S. Francis.
W. H. Soloman.	H. Bacon.	S. E. Pedler.
C. R. Shattock.	E. G. Walton.	R. D. Knight.
C. A. Buckell.	W. E. Griffin.	A. H. Bell.
C. Mills.	S. C. Bowle.	R. J. Green.
A. H. Staple.	G. F. Sargood.	E. Phillips.
H. L. Whitlow.	S. J. Saunders.	F. R. E. Palmer.
P. J. Reid.	J. W. Walton.	H. C. Visick.

DRESSERS IN THE CONSERVATION ROOM.

O. Black.	A. R. Cummings.	H. N. Hillier.
W. W. Iles.	P. H. Furnival.	W. E. Lowe.
G. F. Sargood.	J. F. Rey.	A. H. A. Stevens.
V. R. Searle.	A. H. Clogg.	H. J. Corin.
E. L. Davis.	O. H. Dignum.	R. Edridge.
S. R. Lidiard.	G. H. Morris.	K. C. Ness.
A. H. Smith.	E. Couchman.	H. L. Dent.
J. S. Francis.	H. Hatton.	S. E. Pedler.
V. E. Sams.	H. R. Shapland.	J. L. Wartski.
C. S. Morris.	G. H. Aylen.	A. H. Bell.
A. Drewitt.	A. H. Gabriel.	H. W. Green.
P. Greenwood.	J. H. Hinton.	R. D. Knight.
C. H. Mason.	C. Mills.	A. Archer.
J. Milligan.	W. Morgan.	D. H. Wallis.
E. H. Wyand.	E. G. Smith.	G. W. Badcock.
J. Bennett.	F. W. Corfe.	J. J. Jimenez.
G. F. Knowles.	S. J. Saunders.	C. R. Shattock.
A. H. Staple.	V. E. Turner.	R. S. Witcomb.
A. H. Forbes.	P. J. Reid.	H. B. Ross.
J. E. Spiller.	H. C. Visick.	P. F. Minett.
W. E. Meads.	F. G. Day.	C. A. Buckell.
T. A. Chignell.	W. E. Griffin.	J. W. Powell.
A. C. Stroud.	W. H. Solomon.	H. L. Whitlow.
H. Bacon.	A. R. Beaumont.	J. H. Greenwood.
C. Hickes.	W. K. Perry.	H. S. Wright.
T. Robinson.	H. L. Shelton.	G. H. Steweni.
H. J. Webb.	C. D. Wood.	G. S. H. Barnett.
S. Clifford.	W. Giles.	T. C. Holford.
W. H. Peatfield.	A. B. W. Rust.	A. E. Steele Perkins.
F. R. E. Palmer.	J. B. Barron.	R. J. Green.
H. D. Griffith.	W. Henderson.	E. Phillips.
A. D. Steele Perkins.	L. Webb.	A. W. Aldis.
J. W. Walton.	E. Cock.	E. E. Lacey.
L. Myer.	P. Scott.	J. B. Ball.
H. J. Coish.	F. H. Lennox Jones.	H. Thacker.
J. Cameron.	J. A. Donald.	W. C. Lyne.
J. S. Shoveller.	W. Reynolds.	H. S. Cranston.
C. J. Lamb.	C. J. Pellow.	E. White.
H. W. Wallis.		

JUNIOR DRESSERS IN THE CONSERVATION ROOM.

C. A. W. Buckell.	W. E. Griffin.	C. Mills.
H. L. Whitlow.	R. J. Green.	P. J. Reid.
A. C. Stroud.	J. W. Walton.	A. H. Staple.
A. H. Bell.	T. A. Chignell.	E. Phillips.
E. Cock.	F. R. E. Palmer.	A. H. Forbes.
W. Giles.	H. D. Griffith.	W. Henderson.
A. E. Steele Perkins.	W. H. Peatfield.	C. D. Wood.
E. H. Wyand.	H. C. Visick.	H. T. Binns.
J. Cameron.	N. James.	H. W. Wallis.
J. A. Donald.	J. S. Shoveller.	H. J. Coish.
H. Thacker.	E. E. Lacey.	C. J. Lamb.
W. C. Lyne.	E. H. Mellin.	P. Scott.
H. S. Cranston.	C. J. Pellow.	W. Reynolds.
W. S. Stevens.	A. L. Moon.	H. Croot.
H. P. Aubrey.	R. W. Jones.	A. Goodey.
T. Vernon.		

LIST  
OF  
GENTLEMEN EDUCATED AT GUY'S HOSPITAL  
WHO HAVE PASSED THE  
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES  
&c., &c.,  
IN THE YEAR 1901.

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**University of Oxford.**

*Degree of Doctor of Medicine.*

A. A. H. PARTRIDGE.

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**University of Cambridge.**

*Degree of Doctor of Medicine.*

A. J. COLLIS.      |      S. E. DENYER.      |      A. E. PORTER.

*Final Examination for the Medical and Surgical Degrees.*

Part I.

J. A. Andrews.	G. S. Graham-Smith.	F. Richmond.
E. Bigg.	E. F. G. T. Heap.	H. Wachter.
E. I. Claxton.	F. B. Manser.	J. A. Wood.
H. Davies-Colley.	F. H. Parker.	

Part II.

J. M. Brydone.	J. A. Glover.	R. D. Smedley.
E. I. Claxton.	M. C. Hayward.	N. F. Ticehurst.
C. H. Glenn.	J. S. S. Perkins.	E. G. Wales.

*Second Examination for the Medical and Surgical Degrees.*

H. A. Cutler.	Part I.	H. P. Wiltshire.
	Part II.	
E. C. Hughes.	B. H. Stewart.	F. L. Woods.

*Examination in Sanitary Science.*

Parts I. and II.		
J. B. Anderson.	H. De R. Morgan.	H. J. Starling.
K. W. Goadby.	S. T. Reid.	D. P. Watson.

University of London.

*Examination for the Degree of Doctor of Medicine.*

<p style="text-align: center;">W. H. M. Telling.  <i>Obtained the Gold Medal.</i></p>		
<p>J. T. Dunston.  H. L. Eason.</p>	<p>W. N. East.  J. Moore.</p>	<p>F. S. Lloyd.  (State Medicine).</p>

*Examination for the Degree of Master in Surgery.*

**P. Turner.**

*Examination for the Degree of Bachelor of Surgery.*

Second Division.	
K. B. Alexander.	D. W. Smith.
H. C. Keates.	K. V. Trubshaw.

*Examination for the Degree of Bachelor of Medicine.*

May.		
First Division.		
G. Clarke.		D. G. Greenfield.
T. H. B. Dobson.		J. A. B. Hammond.
Second Division.		
G. T. Collins.		H. B. Foster.
		E. T. Jensen.
October.		
First Division.		
H. C. Keates.		

*Obtained First Class Honours in Medicine.*

**J. F. Northcott.**  
*Obtained Honours in Obstetric Medicine.*

J. Atkins.	W. M. Robson.	K. V. Trubshaw.
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Second Division.

W. H. Bowen.

*Obtained Honours in Medicine.*

M. Coplans.

*Obtained Honours in Medicine.*

K. B. Alexander.	W. G. Parker.	C. Tessier.
R. Balderston.	W. B. Secretan.	A. J. Wernet.
H. W. Brown.	D. W. Smith.	F. C. Wetherell.
B. W. Moss.	L. E. Stamm.	

*Intermediate Examination in Medicine.*

January.

Entire Examination.

Second Division.

P. A. Peall.	H. D. Smart.	H. Watts.
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*Excluding Physiology.*

Second Division.

W. H. Cole.	C. H. Dawe.
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*Physiology only.*

Second Division.

H. M. Goldstein.	H. S. Jones.
C. D. Pye-Smith.	E. W. Strange.

July.

Honours Examination.

A. M. Webber.

*Obtained First Class Honours in Physiology and Histology, and Honours in Anatomy.*

G. W. Russell.

*Obtained Honours in Physiology and Histology, and in Materia Medica and Pharmaceutical Chemistry.*

M. G. Louisson.	H. F. B. Walker.
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*Obtained Honours in Physiology and Histology.*

Entire Examination.

First Division.

H. H. Carter.	H. C. C. Mann.
J. H. Clatworthy.	G. W. Smith.

Second Division.

C. E. Iredell.	B. Moiser.
A. E. F. Kynaston.	F. Rogerson.



*Excluding Physiology.*

First Division.

G. H. Rees.

*Physiology only.*

Second Division.

C. H. Dawe.

|

H. K. Lacey.

*Preliminary Scientific (M.B.) Examination.*

January.

Entire Examination.

First Division.

C. C. A. De Villiers.

|

W. H. Miller.

|

F. T. H. Wood.

Second Division.

T. C. Pocock.

*Chemistry and Experimental Physics only.*

T. H. Barton.

|

P. D. F. Magowan.

E. M. Harrison.

E. L. R. Norton.

*Biology only.*

H. S. Knight.

|

F. A. Sharpe.

E. F. Milton.

J. T. Smalley.

July.

Entire Examination.

First Division.

J. S. Bookless.

|

G. Cockcroft.

H. O. Brookhouse.

H. F. Vandermin.

Second Division.

E. Alban.

|

J. E. Hodson.

|

P. F. McEvedy.

A. W. Berry.

T. B. Layton.

*Chemistry and Experimental Physics only.*

S. W. Daw.

|

E. C. Lowe.

|

A. Zorab.

A. L. Foster.

St. J. A. M. Tolhurst.

*Biology only.*

C. A. Basker.

|

J. A. Bullbrook.

*Intermediate Examination in Science.*

Second Division.

F. W. Hogarth.

*Honours Candidate recommended for a Pass.*

W. H. Dencer.

*Intermediate Examination in Science and Preliminary Scientific Examination conjointly.*

### Examination for Honours.

**B. J. Smart.**

**F. T. H. Wood.**

*Obtained Honours in Chemistry.*

**W. H. Miller.**

*Obtained Honours in Chemistry and Experimental Physics.*

University of Durham.

*Examination for the Degree of Doctor of Medicine.*

H. W. Dudgeon.

F. W. Rowland.

V. Pendred.

T. J. Tulk-Hart.

*Examination for the Degree of Doctor of Medicine for  
Practitioners of Fifteen Years' standing.*

**W. R. Etches.**

**E. H. Graham.**

E. G. Hunt.

*Examinations for the Degrees of Bachelor of Medicine and Surgery.*

### Third Examination.

**B. Glendining.**

**M. C. Wetherell.**

### Second Examination.

J. G. O. H. Lane.

*Obtained First Class Honours.*

### First Examination.

J. G. O. H. Lane.

Victoria University.

August, 1901.

*Examination for the Degrees of Bachelor of Medicine and  
of Surgery.*

A. R. Thompson.

**Royal College of Physicians of Edinburgh.**

*Examination for the Diploma of Membership.*

G. N. Meachen.

**Royal College of Physicians of London.**

*Elected to the Fellowship.*

W. B. Beatson  
J. H. Bryant.

G. F. Still.  
W. J. Tyson.

*Examination for the Diploma of Membership.*

F. R. B. Bisshopp.

*Examination for the Diploma in Public Health.*

T. Halliwell.

A. H. Spicer.

*Final Examination for the License.*

**January.**

R. D. Attwood.  
T. R. Beale-Brown.  
F. G. Cross.  
F. Curtis.  
J. A. B. Hammond.

E. A. Longhurst.  
L. Pern.  
J. F. Robinson.  
E. W. H. Shenton.  
G. Shorland.

A. Ayre Smith.  
D. W. Smith.  
A. W. Talbot.  
R. Tilbury.

**April.**

J. Atkins.  
E. C. Bevers.  
T. H. Body.  
A. E. Cawston.  
G. G. Davidson.  
D. G. Greenfield.  
G. S. Graham-Smith.

E. F. G. T. Heap.  
L. Hirsch.  
P. S. Mandy.  
E. T. Jensen.  
T. A. Matthews.  
D. L. Morgan.  
S. J. Ormond.

E. Shelton-Jones.  
N. F. Ticehurst.  
K. V. Trubshaw.  
E. G. Wales.  
P. H. Ward.  
F. D. Welch.  
G. T. Willan.

**July.**

H. V. Bagshawe.  
W. H. Brailey.  
M. Abdy Collins.  
J. N. Dyson.  
A. C. H. Gray.  
E. J. F. Hardenberg.

B. Instone.  
E. H. Kitchin.  
A. C. Lewis.  
G. H. H. Manfield.  
B. P. O'Neill.  
W. M. Robson.

R. S. Roper.  
E. Stott.  
A. R. Thompson.  
H. Wachter.  
A. Wylie.

**October.**

E. G. Andrew.  
J. A. Andrews.  
S. C. H. Bent.  
W. H. Bowen.  
H. A. Ehrlich.  
H. K. Lacey.

F. B. Manser.  
R. P. Marshall.  
E. J. O'Meara.  
F. M. M. Ommanney.  
A. Pearson.  
A. E. H. Pakes.

A. W. Soper.  
J. B. A. Treusch.  
J. A. Wood.  
M. D. Wood.

**Royal College of Surgeons of Edinburgh.**

*Examination for the Fellowship.*

A. Kinsey-Morgan.

**Royal College of Surgeons of England.**

*Final Examination for the Fellowship.*

C. Banting.	W. R. Nichol.	P. Turner.
F. S. Batchelor.	R. P. Rowlands.	P. N. Vellacott.
H. W. Bruce.	W. B. Secretan.	L. Wilkin.
A. R. McLachlan.	G. S. Simpson.	

*First Examination for the Fellowship.*

K. Black.	E. H. B. Milsom.	A. R. Thompson.
G. Carlisle.	R. H. J. Swan.	P. Turner.
P. P. Cole.	N. Ivens Spriggs.	A. M. Webber.

*Final Examination for the Membership.*

**January.**

R. D. Attwood.	E. A. Longhurst.	A. Ayre Smith.
T. R. Beale-Brown.	L. Pern.	D. W. Smith.
F. G. Cross.	J. F. Robinson.	A. W. Talbot.
F. Curtis.	E. W. H. Shenton.	R. Tilbury.
J. A. B. Hammond.	G. Shorland.	

**April.**

J. Atkins.	E. F. G. T. Heap.	E. Shelton-Jones.
E. C. Bevers.	L. Hirsch.	N. F. Ticehurst.
T. H. Body.	P. S. Mandy.	K. V. Trubshaw.
A. E. Cawston.	E. T. Jensen.	E. G. Wales.
G. G. Davidson.	T. A. Matthews.	P. H. Ward.
D. G. Greenfield.	D. L. Morgan.	F. D. Welch.
G. S. Graham-Smith.	S. J. Ormond.	G. T. Willan.

**July.**

H. V. Bagshawe.	B. Instone.	R. S. Roper.
W. H. Brailley.	E. H. Kitchin.	E. Stott.
M. Abdy Collins.	A. C. Lewis.	A. R. Thompson.
J. N. Dyson.	G. H. H. Manfield.	H. Wachter.
A. C. H. Gray.	B. P. O'Neill.	A. Wylie.
E. J. F. Hardenberg.	W. M. Robson.	

**October.**

E. G. Andrew.	F. B. Manser.	A. W. Soper.
J. A. Andrews.	R. P. Marshall.	J. B. A. Treusch.
S. C. H. Bent.	E. J. O'Meara.	J. A. Wood.
W. H. Bowen.	F. M. M. Ommannney.	M. D. Wood.
H. A. Ehrlich.	A. Pearson.	
H. K. Lacey.	A. E. H. Pakes.	

*Final Examination for the License in Dental Surgery.*

May.

G. W. Badcock.	W. E. Lowe.	N. P. Shepherd.
A. H. Clogg.	R. D. Knight.	E. G. Smith.
H. J. Corin.	G. H. Morris.	A. H. Staple.
E. L. Davis.	A. W. Penrose.	G. H. Steweni.
E. J. Gaffney.	T. Robinson.	V. K. Turner.
J. H. Hinton.	G. F. Sargood.	

November.

G. S. H. Barnett.	R. J. Green.	P. J. Reid.
J. B. Barron.	W. E. Griffin.	J. E. Spiller.
A. H. Bell.	E. A. Longhurst.	H. C. Visick.
C. A. W. Buckell.	C. Mills.	H. L. Whitlow.
S. Clifford.	F. R. E. Palmer.	C. D. Wood.
F. G. Day.	W. H. Peatfield.	E. H. Wyand.
J. S. Francis.	E. Phillips.	

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**Society of Apothecaries of London.**

*Final Examination for the License.*

F. A. Beattie.	E. S. Perkins.	F. A. Segreda.
C. A. Lower.		

## MEDALLISTS AND PRIZEMEN.

JULY, 1902.

*Open Scholarships in Arts.*

Clifford Antony Leo Mayer, Royal College of Mauritius, £100.

Malcolm John Rattray, King's School, Bruton, £50.

Thomas Edmund Ashdown Carr, Lancing's College, Certificate.

*Open Scholarships in Science.*

Percy Strickland Mills, Dulwich College, £105.

William Henry Trethowan, Technical School, Plymouth, £105.

Charles Morley Wenyon, University College, Certificate.

*Scholarship for University Students.*

Arthur Frederick Hertz, Magdalen College, Oxford, £50.

William Mayhew Mollison, King's College, Cambridge, Certificate.

Ernest Cranmer Hughes, Clare College, Cambridge, Certificate.

*Senior Proficiency Prizes.*

Claude Tessier, Certificate.

*Junior Proficiency Prizes.*

Frank Thomas Herbert Wood, £20.

Frederick Harold Lennox-Jones

Arthur Boniface O'Brien

Morris de Lancey Robinson

} equal £8 6s. 8d. each.

Alfred Gwilym Jones, Certificate.

Richard William Allen, Certificate.

Herbert Andrew Watney, Certificate.

*The Michael Harris Prize for Anatomy.*

Arthur Boniface O'Brien, £10.

Alfred Gwilym Jones, Certificate.

*The Wooldridge Memorial Prize for Physiology.*

Richard William Allen

Morris de Lancey Robinson

Frank Thomas Herbert Wood

Walter Welchman, Certificate.

Herbert Andrew Watney, Certificate.

} equal £3 6s. 8d.

*The Hilton Prize for Dissections (1901).*

Edward Crosby Peers, £2.

John Edmund Spiller, £2.

John Alan Campbell Greene, £1.

*The Arthur Durham Prizes for Dissection.*

*First Year's Students.*

William Henry Trethowan, £5.

Stanley Arthur Piper, Certificate.

*Senior Students.*

Richard William Allen, £15.

Frank Thomas Herbert Wood, Certificate.

*The Treasurer's Gold Medal for Clinical Medicine.*

Carrick Hey Robertson.

*The Treasurer's Gold Medal for Clinical Surgery.*

William Henry Bowen.

*The Beane Prize in Pathology (1901).*

Herbert Stanley French, £34.

*The Golding-Bird Gold Medal and Scholarship in Sanitary  
Science.*

Burton Alexander Nicol, £20.

## THE PHYSICAL SOCIETY.

**Honorary President.**—Sir SAMUEL WILKS, Bart., M.D., LL.D., F.R.S.

**Secretaries.**—Mr. Bellingham Smith and Dr. Beddard.

### Presidents.

F. G. Cross, S. Hodgson, M.B., G. G. Davidson, B.A., F. W. Morton-Palmer, B.A., P. N. Blake-Odgers, B.A., D. G. Greenfield, M.B., C. H. Robertson, P. R. Bolus, H. F. B. Walker, H. Barber, M.B., A. C. H. Gray, Owen W. Richards, M.A.

### PRIZEMEN FOR THE SESSION 1901-1902.

The Society's Prize of £10 was awarded to Mr. Owen Richards, M.A., for his paper, "Medical Heresies," and Mr. P. N. Blake Odgers, B.A., gained the Treasurer's Prize of £5 for his paper on "Traumatic Fever."

## CLINICAL APPOINTMENTS HELD DURING THE YEAR 1901.

### HOUSE PHYSICIANS.

E. W. Goble.	A. H. Davies.	L. E. Stamm.
T. E. Holmes.	J. A. Butler.	H. S. French.
F. G. Gibson.	D. Forsyth.	

### HOUSE SURGEONS.

T. P. Thomas.	W. B. Secretan.	P. W. L. Camps.
J. S. Steele Perkins.	F. G. Cross.	F. Curtis.
C. H. Glenn.	H. McD. Parrott.	

### ASSISTANT HOUSE SURGEONS.

J. S. Steele Perkins.	A. Densham.	T. J. Wright.
F. W. Sime.	P. W. L. Camps.	H. McD. Parrott.
F. O. Stoehr.	D. W. Smith.	G. Lewin.
F. G. Gibson.	F. Curtis.	F. G. Cross.
E. F. G. T. Heaps.	J. A. B. Hammond.	P. C. P. Ingram.
S. J. Ormond.	C. H. Glenn.	E. C. Bevers.
M. A. Collins.	W. H. Brailey.	D. G. Greenfield.
T. H. B. Dobson.	N. F. Ticehurst.	J. A. Glover.

### RESIDENT OBSTETRIC ASSISTANTS.

C. B. Thompson.	C. H. Brangwin.	J. F. Northcott.
B. W. Moss.	F. W. Sime.	D. P. Watson.
T. J. Wright.	H. Wachter.	T. P. Thomas.
J. M. Brydone.		



CLINICAL ASSISTANTS.

P. T. Manson.	F. G. Gibson.	J. A. Butler.
H. S. French.	N. F. Ticehurst.	F. Curtis.
J. A. Andrews.	E. C. Bevers.	E. F. G. T. Heap.
D. L. Morgan.	S. J. Ormond.	J. F. Robinson.
D. G. Greenfield.	M. A. Collins.	A. O. H. Gray.
J. A. Glover.	G. Clarke.	T. H. B. Dobson.
A. Wylie.	H. K. Lacey.	E. I. Claxton.
A. C. Ransford.	A. Pearson.	G. G. Davidson.

CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

G. G. Davidson.	M. J. Rees.	E. H. Kitchen.
T. H. B. Dobson.	J. Atkins.	H. Davies-Colley.
H. K. Lacey.	E. I. Claxton.	C. E. Gaitskell.
A. D. E. Kennard.	F. B. Manser.	A. W. Soper.
G. B. Churchill.	A. H. E. Wall.	G. F. Humphreys.
F. L. Thomas.		

CLINICAL ASSISTANTS IN THE SURGICAL WARDS.

E. Bigg.	D. S. Graves.	W. G. Parker.
R. D. Smedley.	W. H. Cole.	A. J. Beadel.
H. A. Gaitskell.	W. O. Roberts.	G. W. Smith.

SURGEONS' DRESSERS.

F. M. M. Ommanney.	R. G. Anderson.	G. Moir.
A. H. Turner.	H. J. Gater.	A. J. Urquhart.
A. C. Osburn.	G. E. Malcomson.	A. H. E. Wall.
H. Barber.	A. H. E. Pakes.	A. J. Beadel.
B. Churchill.	J. C. Curtis.	N. N. Houghton.
W. C. C. Jones.	T. G. Miles.	W. A. G. Stevens.
W. H. Bowen.	M. W. Cohen.	F. H. Parker.
F. L. Thomas.	P. N. B. Odgers.	M. C. Wetherell.
G. B. S. Soper.	G. W. C. Hollist.	O. B. Travers.
F. J. Turner.	F. M. V. Smith.	G. T. Wrench.
E. Faulks.	H. T. Palmer.	T. M. Smith.
L. G. Nash.	W. H. Cole.	C. J. Pinching.
A. W. Iredell.	J. D. Pearson.	C. M. L. Cowper.
F. L. Thomas.	O. W. Richards.	K. Anderson.
C. H. Denyer.	A. P. Piggot.	J. B. Copland.
H. R. Grellet.	G. S. Robertson.	E. H. Griffin.
C. H. Robertson.	H. Tipping.	W. L. M. Day.
L. H. Moiser.	C. M. Murray.	H. S. Jones.
S. L. Pallant.	W. E. J. Tuohy.	C. F. Fraser.
G. W. Smith.	J. Braithwaite.	L. S. H. Glanville.
W. W. Read.	J. Bickerton.	E. W. Strange.
W. F. Box.	P. C. V. Bent.	H. S. Brown.
E. G. Goldie.	J. H. Donnell.	F. W. Fawcsett.
B. Glendining.	B. H. Wedd.	
P. W. Hamond.	H. M. Goldstein.	

## ASSISTANT SURGEONS' DRESSERS.

G. T. Wrench.	J. S. Cooper.	H. L. Shelton.
O. B. Travers.	G. W. C. Hollist.	S. L. Prall.
H. T. Palmer.	R. H. Terry.	E. Faulks.
O. W. Richards.	J. D. Pearson.	C. J. Pinching.
M. C. Wetherell.	A. W. Iredell.	T. M. Smith.
C. E. Bartlett.	E. J. Gaffney.	N. I. Spriggs.
H. S. Brown.	L. G. Nash.	F. J. Turner.
C. M. L. Cowper.	W. H. Cole.	G. S. Robertson.
J. Goss.	A. P. Piggot.	B. I. Rahim.
G. W. Smith.	J. B. Copland.	C. M. Murray.
H. R. Grellet.	B. Glendining.	C. H. Denyer.
S. L. Pallant.	W. E. J. Tuohy.	K. Anderson.
C. H. Robertson.	E. H. Griffin.	W. L. M. Day.
L. H. Moiser.	H. Tipping.	C. F. Fraser.
E. N. Jupp.	W. H. Bush.	H. S. Jones.
W. F. Box.	C. M. Anthony.	P. C. V. Bent.
J. H. Donnell.	B. H. Wedd.	E. W. Strange.
B. B. Westlako.	W. W. Read.	H. M. Goldstein.
R. E. Brayne.	J. Braithwaite.	T. Morgan.
P. W. Hamond.	G. L. Buckeridge.	F. W. Fawcsett.
L. S. H. Glanville.	J. M. Bickerton.	R. G. Anderson.
J. T. Hicks.	E. G. Goldie.	F. C. Knight.
H. E. Morris.	H. Johnson.	C. R. Shattock.
T. C. Lucas.	C. H. Bubb.	F. C. Robinson.
H. B. German.	W. M. Woodward.	D. R. Pike.
A. R. Brailey.	H. Ackroyd.	C. E. Adams.
G. F. Hardy.	A. R. Wilson.	H. C. Winckworth.

## DENTAL SURGEONS' DRESSERS.

G. T. Collins.	F. W. Sime.	M. J. Rees.
C. H. Glenn.	A. Wylie.	C. Tessier.
H. Bentley.	J. Evans.	F. A. Beattie.
R. C. Lawry.	W. C. Lewis.	H. J. Gater.
D. L. Morgan.		

## CLINICAL ASSISTANTS IN MEDICAL OUT-PATIENTS.

E. G. Wales.	C. Tessier.	H. W. Brown.
E. Stott.	H. Wachter.	T. T. Kelly.
E. J. F. Hardenberg.	F. W. Smith.	W. H. Bowen.
M. Coplans.	A. E. H. Pakes.	A. Wylie.
T. G. Miles.	G. Evans.	A. C. Osburn.
E. H. Kitchen.		

## DRESSERS IN THE EYE WARDS.

K. V. Trubshaw.	R. Thompson.	E. J. F. Hardenberg.
F. B. Manser.	W. J. Davies.	M. D. Wood.
W. W. C. Jones.	E. P. Mitchell.	J. Evans.
F. M. M. Ommanney.	J. W. Gromitt.	A. J. Urquhart.
T. T. Kelly.	R. C. Lawry.	G. F. Humphreys.
R. T. Collins.	H. C. Keates.	J. C. O. Bradbury.
R. D. Smedley.	F. H. Parker.	J. A. Andrews.
A. W. Gater.	H. Barber.	D. L. Morgan.
E. H. Griffin.	T. H. Roberts.	W. A. G. Stevens.
H. J. Gater.	D. R. T. Griffiths.	

**DRESSERS IN THE THROAT DEPARTMENT.**

J. D. Bridger.	J. A. B. Hammond.	A. W. Gater.
J. A. Wood.	H. Davies-Colley.	J. A. Andrews.
G. T. Collins.	G. Evans.	W. J. Davies.
R. P. Marshall.	R. S. Roper.	W. M. Robson.
D. H. Trail.	F. E. Welchman.	J. Evans.
C. Tessier.	R. Thompson.	J. C. O. Bradbury
J. T. Dunston.	H. C. Keates.	M. J. Rees.
A. H. E. Wall.	R. C. Lawry.	H. Barber.

**CLERKS IN THROAT DEPARTMENT.**

R. P. Marshall.	D. W. Smith.	S. C. Bowle.
J. Goss.	G. G. Davidson.	B. I. Rahim.
P. P. Cole.	G. Moir.	

**MEDICAL WARD CLERKS.**

K. Anderson.	C. H. Robertson.	W. E. J. Tuohy.
J. B. Copland.	J. T. Hicks.	H. L. Shelton.
G. W. Smith.	H. Tipping.	W. L. M. Day.
B. Glendining.	E. H. Griffin.	L. H. Moiser.
B. B. Westlake.	J. Braithwaite.	C. H. Bubb.
E. W. Strange.	P. W. Hamond.	C. E. Adams.
W. F. Box.	H. M. Goldstein.	B. H. Wedd.
P. C. V. Bent.	J. M. Bickerton.	R. E. Brayne.
G. L. Buckeridge.	W. H. Bush.	J. H. Donnell.
F. W. Fawcett.	C. E. Fraser.	L. S. Glanville.
E. G. Goldie.	E. W. Jupp.	W. W. Read.
R. G. Anderson.	C. M. Anthony.	H. S. Jones.
B. B. Westlake.	F. C. Robinson.	A. R. Brailey.
H. C. Winckworth.	H. Ackroyd.	J. S. Cooper.
E. J. Gaffney.	D. R. Pike.	C. R. Shattock.
H. S. Brown.	H. B. German.	F. C. R. Knight.
T. C. Lucas.	H. Johnson.	H. E. Morris.
A. R. Wilson.	H. M. Woodward.	G. F. Hardy.
D. H. Richards.	C. H. Dawe.	J. Bromley.
R. Moyle.	A. E. Rowlett.	H. W. Bethell.
R. Larkin.	C. H. Reinhold.	H. D. Smart.
N. I. Spriggs.	H. Watts.	F. H. Wallace.
J. Goss.	F. L. Ward.	F. P. Hughes.
J. W. Dadd.	P. R. Bolus.	P. A. Peall.
C. E. Iredell.	H. C. C. Mann.	R. Willan.
C. D. Pye-Smith.	M. B. Taylor.	K. Black.
H. O. M. Beadnell.	C. S. Morris.	F. G. Goble.
S. C. Bowle.	G. Carlisle.	J. F. Rey.
B. W. Lacey.	E. H. B. Milsom.	H. Watts.
W. T. Meade-King.	B. I. Rahim.	

**ASSISTANT PHYSICIANS' CLERKS.**

H. R. Grellet.	T. L. Pallant.	G. S. Robertson.
A. P. Piggot.	W. L. M. Day.	C. M. L. Cowper.
C. M. Murray.	H. Tipping.	C. H. Denyer.
C. H. Bubb.	J. Braithwaite.	C. E. Adams.
B. Wedd.	L. H. Glanville.	W. H. Bush.
C. H. Dawe.	E. G. Goldie.	P. W. Hamond.
R. E. Brayne.	P. C. V. Bent.	E. J. Gaffney.
A. R. Brailey.	D. R. Pike.	C. R. Shattock.
H. S. Brown.	H. B. German.	H. C. Winckworth.
H. M. Woodward.	T. C. Lucas.	J. S. Cooper.
D. H. Richards.	H. W. Bethell.	F. H. Wallace.
E. L. Ward.		

## SURGICAL WARD CLERKS.

F. C. R. Knight.	F. C. Robinson.	G. F. Hardy.
H. Johnson.	H. B. German.	C. R. Shattock.
H. C. Winckworth.	H. Ackroyd.	A. R. Brailey.
J. S. Cooper.	T. C. Lucas.	D. R. Pike.
J. Goss.	D. H. Richards.	B. I. Rahim.
A. R. Wilson.	H. M. Woodward.	J. Bromley.
M. B. Taylor.	H. W. Bethell.	C. H. Reinhold.
H. D. Smart.	H. Watts.	F. H. Wallace.
R. Moyle.	A. E. Rowlett.	P. R. Bolus.
F. P. Hughes.	C. E. Iredell.	P. A. Peall.
C. D. Pye-Smith	H. O. M. Beadnell.	K. Black.
S. C. Bowle.	G. Carlisle.	F. G. Goble.
B. W. Lacey.	H. C. C. Mann.	E. H. B. Milsom.
C. S. Morris.	J. F. Rey.	W. P. Mead-King.
P. P. Cole.	J. W. Dadd.	A. M. Webber.
R. G. Seagrove.	F. B. Lowe.	H. H. Carter.
A. E. Kynaston.	M. G. Louisson.	G. H. Rees.
G. Russell.	B. Moiser.	M. J. Mottram.
R. P. Rowlands.	B. H. Stewart.	H. H. Jenkins.
G. C. F. Robinson.	H. F. B. Walker.	L. H. Frankenberg.
G. A. Ticehurst.	E. Lloyd.	R. A. Greeves.
L. J. Orpen.	O. V. Payne.	

## CLERKS IN THE SKIN DEPARTMENT.

S. J. Ormond.	F. W. Smith.	J. D. Bridger.
E. J. F. Hardenberg.	R. P. Marshall.	A. E. H. Pakes.
W. A. G. Stevens.	J. Goss.	

## AURAL SURGEON'S DRESSERS.

R. S. Roper.	H. Bentley.	G. Clarke.
M. Coplans.	G. H. H. Manfield.	E. Bigg.
R. C. Lawry.	G. T. Collins.	G. S. Graham-Smith.
A. D. E. Kennard.	T. A. Matthews.	A. Wylie.
A. C. Osburn.	M. W. Cohen.	L. E. Stamm.

## ASSISTANT SURGEONS' CLERKS.

H. W. Bethell.	S. C. Bowle.	K. Black.
C. H. Reinhold.	F. H. Wallace.	J. Bromley.
R. Moyle.	D. H. Richards.	B. W. Lacey.
F. G. Goble.	F. Barnes.	H. H. Jenkins.
F. C. Peers.	G. Nunn.	F. W. M. Palmer.
C. P. Harvey.	J. D. Thomas.	R. Franklin.
A. V. Maybury	A. J. Mollison.	M. Hertz.

POST-MORTEM CLERKS.

H. B. Dismorr.	D. R. T. Griffiths.	C. R. Howard.
W. H. Brailey.	M. Coplans.	J. W. Gromitt.
F. M. V. Smith.	H. W. Brown.	R. T. Collins.
A. H. Turner.	H. Wacher.	B. Glendining.
H. Bentley.	J. A. Wood.	W. C. Lewis.
C. M. Murray.	W. W. C. Jones.	F. D. S. Jackson.
G. L. Buckeridge.		

CLERKS IN THE ELECTRICAL DEPARTMENT.

M. Coplans.	J. F. Robinson.	F. D. Welch.
S. E. Denyer.	A. C. Osburn.	R. P. Marshall.
M. W. Cohen.	G. G. Davidson.	

OBSTETRIC DRESSERS.

G. Evans.	H. K. Lacey.	R. C. Lawry.
G. Clarke.	H. P. Wiltshire.	H. C. Keates.
P. S. Mandy.	J. Evans.	A. C. Lewis.
G. H. H. Manfield.	F. E. Welchman.	M. Coplans.
F. A. Beattie.	D. H. Trail.	F. C. Wetherell.
H. W. Brown.	G. B. Churchill.	T. G. Miles.
H. A. Cutler.	J. A. Andrews.	A. H. E. Wall.
H. Barber.	A. H. Turner.	J. W. Gromitt.
D. R. T. Griffiths.	G. Moir.	R. T. Collins.
O. T. Travers.	W. C. Lewis.	F. M. V. Smith.
T. M. Smith.	L. H. Moiser.	

EXTERN OBSTETRIC ATTENDANTS.

G. T. Wrench.	F. C. Wetherell.	M. A. Collins.
A. C. H. Gray.	G. Tessier.	D. H. Trail.
D. R. T. Griffiths.	G. Evans.	C. E. Gaitskell.
E. G. Allport.	R. C. Lawry.	H. A. Cutler.
T. F. H. Roberts.	H. W. Brown.	W. M. Robson.
A. Wylie.	H. A. Ehrlich.	G. H. H. Mansfield.
A. C. Ransford.	A. D. E. Kennard.	W. H. Bowen.
A. E. H. Pakes.	W. G. Parker.	G. F. Humphreys.
F. D. S. Jackson.	S. Child.	H. J. Gater.
G. Moir.	W. A. G. Stevens.	T. G. Miles.
M. Coplans.	F. Richmond.	M. W. Cohen.
A. H. E. Wall.	H. P. Wiltshire.	H. Barber.
A. Pearson.	A. C. Osburn.	F. H. Parker.
F. L. Thomas.	P. N. B. Odgers.	G. H. Gask.
T. M. Smith.	N. N. A. Houghton.	C. R. Howard.
G. E. Malcomson.	R. T. Collins.	W. W. C. Jones.
G. B. Churchill.	J. F. Douse.	L. G. Nash.
G. W. Hollist.	E. Roberts.	J. W. Gromitt.
G. B. Soper.	O. W. Richards.	E. Faulks.
H. T. Palmer.	J. E. O. Bradbury.	A. W. Iredell.
C. J. Pinching.	W. H. Cole.	W. E. Tuohy.
W. C. Lewis.	M. C. Wetherell.	J. D. Pearson.
C. M. Murray.	C. E. Bartlett.	C. W. Denyer.
H. L. Shelton.	P. C. Knight.	F. M. V. Smith.
J. Goss.	G. P. C. Hayes.	F. A. Beattie.
C. B. Penny.	E. Bigg.	R. D. Smedley.
A. W. Soper.		

## CLERKS TO ANÆSTHETISTS.

A. E. H. Pakes.	P. T. Manson.	G. Tessier
A. W. Gater.	W. M. Thomas.	A. J. Urquhart.
M. A. Collins.	A. Wylie.	G. T. Willan.
M. Coplans.	W. J. Davies.	J. A. Andrews.
F. G. Heap.	E. I. Claxton.	S. Child.
O. W. Richards.	E. Faulks.	H. T. Palmer.
C. H. Gask.	H. V. Bagshawe.	D. H. Trail.
J. W. Gromitt.	F. Richmond.	R. Jimenez.
J. E. Collins.	A. D. E. Kennard.	G. W. Hollist.
W. G. Parker.	H. P. Wiltshire.	E. Bigg.
G. W. Smith.	E. J. F. Hardenberg.	A. C. H. Gray.
K. Anderson.	R. T. Collins.	T. A. Matthews.
J. A. Glover.	E. H. Griffin.	R. D. Smedley.
J. B. Copland.	G. S. Robertson.	E. Roberts.
E. G. Allport.	W. W. C. Jones.	F. B. Manser.
G. Moir.	A. Pearson.	M. D. Wood.
H. L. Shelton.	J. M. Bickerton.	B. Glendinning.
P. P. Cole.	W. H. Bowen.	W. H. Cole.
T. M. Smith.	F. M. V. Smith.	T. G. Miles.
H. Tipping.	E. H. Kitchen.	G. Evans.
L. H. Moiser.	F. C. Wetherell.	A. H. Turner.
F. J. Turner.	H. J. Gater.	P. N. B. Odgers.
C. E. Bartlett.		

## DENTAL SCHOOL.

## APPOINTMENTS HELD IN THE YEAR 1901.

## DENTAL HOUSE SURGEONS.

S. C. Bowle.	E. W. Corfe.	G. W. Badcock.
A. H. Clogg.		

## ASSISTANT DENTAL HOUSE SURGEONS.

E. L. Davis.	W. E. Griffin.	R. J. Green.
H. L. Whitlow.	G. H. Morris.	H. C. Visick.
E. E. Lacey.	J. E. Spiller.	

## DEMONSTRATORS IN THE CONSERVATION ROOM.

J. B. Barron.	A. C. Stroud.	E. Phillips.
J. Cameron.	E. E. Lacey.	P. Scott.
J. A. Donald.	H. Thacker.	H. P. Aubrey.
H. Croot.	A. L. Moon.	L. U. Ransford.

## ASSISTANT DEMONSTRATORS OF DENTAL MICROSCOPY.

G. S. H. Barnett.	W. E. Griffin.	J. E. Spiller.
A. R. Beaumont.		

## ASSISTANT DEMONSTRATORS OF PRACTICAL DENTAL METALLURGY.

C. Mills.	H. L. Whitlow.
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**DRESSERS IN THE GAS ROOM.**

R. J. Green.	S. Clifford.	T. A. Chignell.
H. C. Visick.	F. G. Day.	A. C. Stroud.
J. W. Walton.	J. E. Spiller.	W. Giles.
A. E. Steele Perkins.	G. S. H. Barnett.	A. H. Forbes.
H. D. Griffiths.	E. H. Wyand.	J. B. Barron.
C. D. Wood.	E. A. Longhurst.	P. J. Reid.
W. Henderson.	P. F. Minett.	J. S. Francis
T. C. Holford.	W. P. Shepherd.	P. Scott.
E. E. Lacey.	H. J. Coish.	A. R. Beaumont.
F. R. E. Palmer.	C. D. Wood.	J. Cameron.
W. C. Lyne.	N. James.	J. S. Shoveller.
J. A. Donald.	J. Stevens.	H. T. Binns.
T. H. Griffin.	L. U. Ransford.	H. Croot.
E. Cock.	A. B. Cocker.	E. O. Stevens.
E. G. Walton.	A. L. Moon.	L. Myer.
H. Bacon.	A. L. Mason.	H. P. Aubrey.
W. S. Stevens.	R. S. Witcomb.	A. L. George.
H. S. Cranston.	H. W. Wallis.	W. Reynolds.
C. J. Pellow.	C. J. Lamb.	E. Farrant.
F. N. Fox.	T. Burton.	E. F. Deck.
R. W. Jones.	W. H. Peatfield.	

**DRESSERS IN THE EXTRACTION ROOM.**

N. James.	F. N. Palmer.	W. J. Goodman.
H. Thacker.	W. R. Penford.	A. L. Mathews.
E. E. Lacey.	J. S. Shoveller.	T. Vernon.
H. T. Binns.	E. H. Wyand.	R. Edridge.
H. W. Wallis.	P. Scott.	W. C. Lyne.
H. J. Coish.	J. Cameron.	H. S. Cranston.
T. H. Griffin.	W. W. Vaughan.	J. Stevens.
A. L. Moon.	C. J. Lamb.	F. H. Lennox Jones.
H. Croot.	J. A. Donald.	H. P. Aubrey.
A. Goodey.	R. W. Jones.	E. O. Stephens.
C. J. Pellow.	W. Reynolds.	J. B. Ball.
A. L. George.	E. White.	A. L. Mason.
R. Wallis.	A. B. Cocker.	H. C. Malleson.
W. S. Stevens.	H. E. Chinneck.	G. W. Gwyther.
A. D. Crofts.	C. S. Kliszczewski.	E. Farrant.
R. H. C. Johnson.	H. S. Chandler.	H. J. Cole.
T. J. Green.	F. N. Fox.	W. E. Derriman.
R. G. Harrington.	T. Burton.	N. B. Soper.

**JUNIOR AND CASUALTY DRESSERS.**

T. H. Griffin.	J. G. Morrell.	H. W. Jones.
J. Stevens.	H. S. Chandler.	A. R. Durant.
L. U. Ransford.	E. O. Stevens.	F. N. Fox.
H. W. Gwyther.	N. B. Soper.	T. Burton.
E. Farrant.	H. J. Fox.	R. H. C. Johnson.
W. E. Derriman.	R. G. Harrington.	F. Barkshire.
H. J. Cole.	W. J. Goodman.	A. L. Mathews.
L. H. Pellow.	P. J. Phillips.	R. Wallis.
W. R. Ransford.	H. E. Chinneck.	T. J. Green.

## DRESSERS IN THE CONSERVATION ROOM.

H. P. Aubrey.	H. S. Chandler.	W. R. Ransford.
A. H. Forbes.	R. J. Harrington.	E. F. Deck.
C. Mills.	E. O. Stevens.	R. W. Jones.
L. U. Ransford.	H. J. Cole.	P. Scott.
H. L. Whitlow.	W. Reynolds.	A. B. Cocker.
F. G. Day.	T. Burton.	R. C. Mungal.
P. F. Minett.	F. N. Fox.	L. H. Pellow.
J. S. Shoveller.	A. L. Moon.	W. E. Griffin.
E. White.	P. J. Reid.	E. Phillips.
A. H. Bell.	G. S. H. Barnett.	A. C. Stroud.
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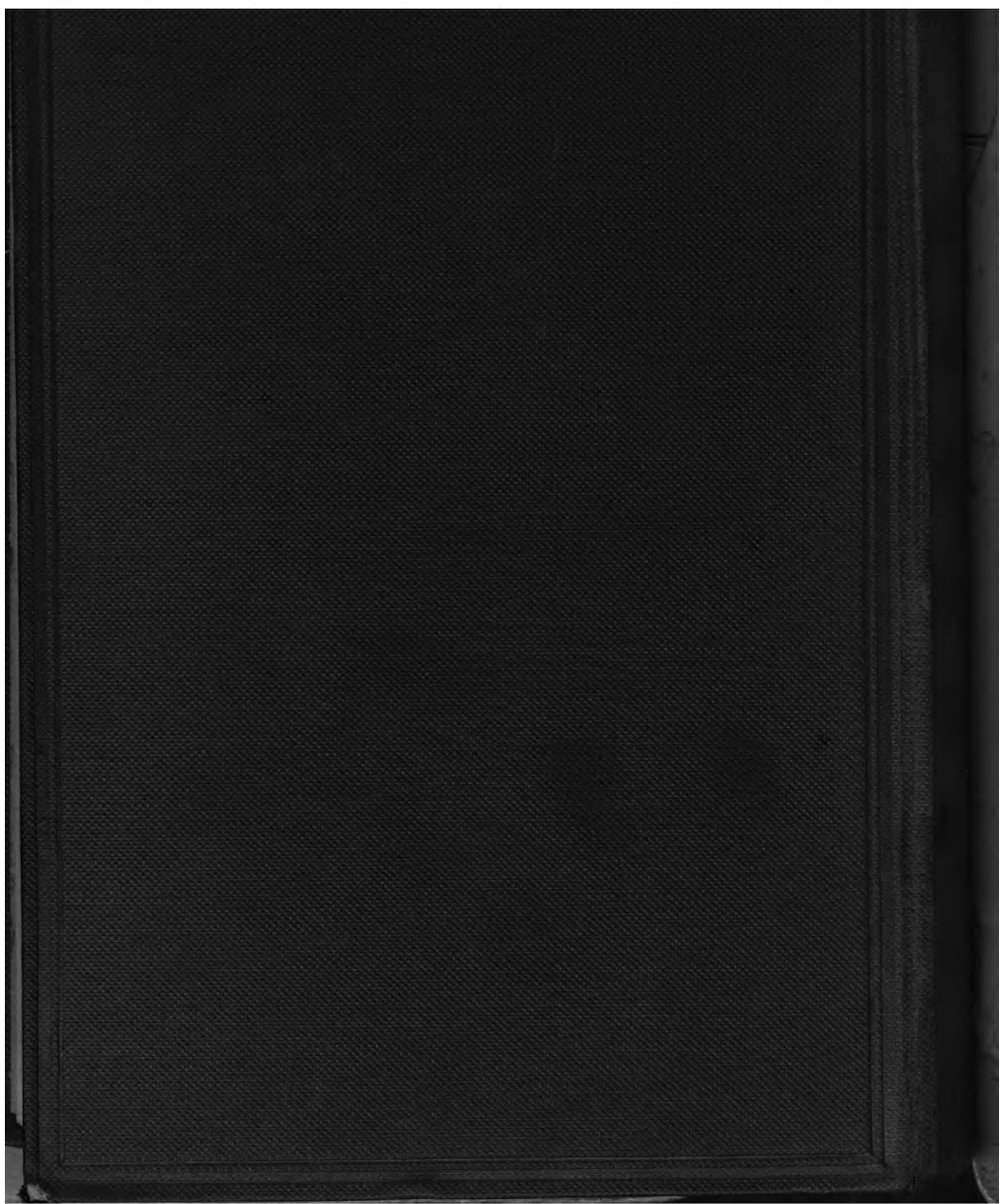


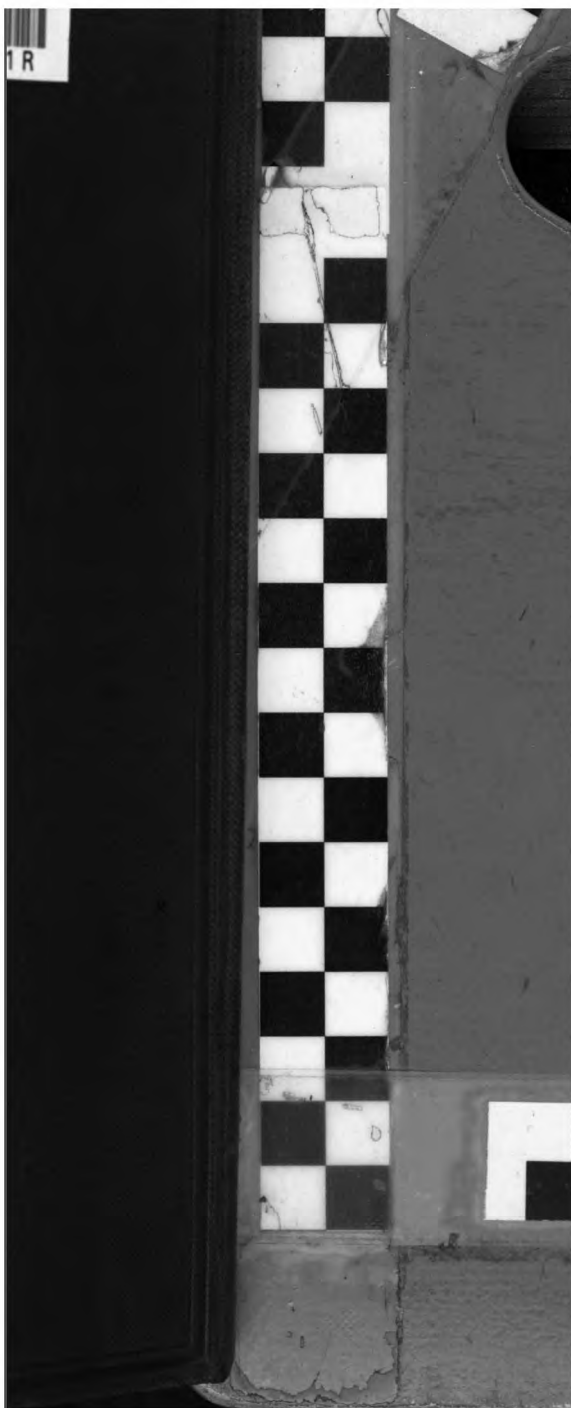












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